A CLINICO PATHOLOGICAL STUDY OF MENINGIOMAS

Dissertation submitted in partial fulfillment of the requirements for the degree of

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CERTIFICATE

This is to certify that this dissertation entitled **"A CLINICO PATHOLOGICAL STUDY OF MENINGIOMAS"** is a bonafide work done by **Dr. RADHA. A.**, in partial fulfillment of the requirements of The TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY, Chennai for the award of M.D. Pathology Degree.

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DECLARATION

I declare that this dissertation entitled **"A CLINICO PATHOLOGICAL STUDY OF MENINGIOMAS"** has been done by me under the guidance and supervision of **Prof. Dr.SHANTHA RAVISANKAR, M.D. DCP.,** It is submitted in partial fulfillment of the requirements for the award of the M.D., Pathology degree by The Tamilnadu Dr.M.G.R. Medical University, Chennai. This has not been submitted by me for the award of any degree or diploma from any other University.

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CONTENTS

INTRODUCTION

"The pathological curiosity of one day becomes in its proper time a common place condition at a later time".

Sir Harvey Cushing

Meningiomas were once considered a complex and diverse group of neoplasms in view of their varied histological patterns. Over the years these morphologically different tumours have been studied in detail and the mysteries of their origins unraveled.

The term "meningioma" was coined by Sir Harvey Cushing, the most revered neurosurgeon of the 20th century. But as early as 1614 Felix Plater removed a brain tumor at autopsy which he described accurately. The neoplasm was in all probability a meningioma. The first brain tumour reported from the United States also happens to be a meningioma.

In 1835, Professor Peccholi, Professor of Surgery and Operating Medicine from the University of Siena operated on a dural based tumour at the occiput which he described as a "fungus of the dura mater". Over a period of 16 years he operated upon and studied the morphology of several such tumours and published a comprehensive review of his work in 1847.

Francesco Durante of Sicily, Italy, was the first surgeon to successfully resect a cranial base meningioma.

But the most celebrated work on meningiomas is by Sir Harvey Cushing co – written by Louise Eisenhardt in 1938 titled "Meningiomas : Their classification, regional behaviour, life history and surgical end results". This works gives incredible details and morphological descripotions of meningiomas.

Various authours over the years have attempted to classify meningiomas. It was understood that most of these tumours followed a benign course and that morbidity and mortality relating to them stem from unusual locations and limitations to surgical resectablity.

Cushing[5] identified 20 subtypes that included non meningothelial mesenchymal tumours too. Further refinement down the year divided meningiomas in to benign, atypical and malignant variants.

The 1993 WHO classification[28] recognizes two broad categories of meningeal neoplasms :

1. Meningiomas arising from meningothelial (arachnoidal cap) cells

2. Tumours arising from mesenchymal non meningothelial cells which are classified as soft tissue tumours.

Kustanikul and Brown[42] attributed the histological diversity of meningiomas to the pluripotential nature of mesenchymal stem cells arising from the neural crest.

AIMS AND OBJECTIVES

1. To study the incidence of intracranial and spinal meningiomas during the five and a half year period from January 2001 to August 2006 at the Institute of Neurology Madras Medical College.

2. To study the distribution of the various sub types of meningiomas with regard to age, sex and site.

3. Histopathological categorization of meningiomas and grading them as per the WHO 2000 classification of Central nervous system tumours.

4. To correlate the histopathological findings with clinical and radiological findings where ever possible.

REVIEW OF LITERATURE

The pachymeninges or duramater and the leptomeninges – the arachnoid membrane and pia mater are generally regarded to be mesodermal in origin. The leptomeninges have a common phylogenetic history, develop embryologically in continuity and preserve this intimate relationship in their final differentiation and hence are regarded together as "pia – arachnoid"[48].

The meninges are composed chiefly of collagen, elastin and reticulin fibres covered by flat cells considered t be mesothelial. The fine arachnoidal trabeculae that traverse the sub – arachnoid space are covered by these mesothelial cells too. These cells have pale cytoplasm with thin long cytoplasmic processes and form several layers.

The cell of origin of meningiomas has long been a matter of hot debate. Endothelial cells, fibroblasts, primitive mesenchymal stem cells and arachnoidal cap cells were considered potential candidates at various points of time. This led to the designation of "meningioma" being extended to diverse neoplasms that shared only the tendency to arise within the histogenetically complex tissues of the leptomeninges or the duramater. Thus dissimilar entitites like meningeal hemangiopericytomas, hemangioblastomas, solitary fibrous tumours and other meningeal sarcomas were once yoked under the term "meningioma"[44].

The combination of electron microscopy and immunhistochemistry with in vitro methodology of cell lines derived form normal leptomeninges and meningiomas has supported the hypothesis that meningiomas are derived from arachnoidal cap cells. These are specialised elements that populate the arachnoid membranes and cap the arachnoid villi associated with intra-dural venous sinuses and their tributaries[48].

CLASSIFICATION OF MENINGIOMAS

Meningiomas present a bewildering variety of histological patterns.

A classical study by Cushing and Eisenhardt[5] demonstrated nine main types and twenty sub types.

Later a working classification identified only four main types: syncytial, fibroblastic, transitional and angioblastic.

Uncertainities regarding the histogeneis of the angioblastic types complicated this classification. The identification of other variants like papillary and rhabadoid types with less favourable prognosis necessitated the formulation of a new classification[39].

In 1993, a new WHO classification was formulated which identified 11 sub-types in addition to papillary, atypical and anaplastic meningiomas.

The latest classification has been the WHO 2000 classification[28], given below[28].

GRADE I

Meningothelial

Fibrous

Transitional

Psammomatous

Angiomatous

Microcystic

Secretory

Metaplastic

Lymphoplasmacytic

GRADE II

Atypical

Clear Cell

Chordoid

GRADE III

Anaplastic / Malignant

Papillary

Rhabdoid

EPIDEMIOLOGY :-

Literature states that the frequency of meningioma is 20% of all intra cranial neoplasms in men and 38% in women. Countries like Africa have a higher frequency at 30%[9] in men.

In India meningiomas are the second largest category of brain tumours after gliomas.

A two year report of the population based cancer registries (1999-2000) by the Indian council of Medical Research states that in Chennai central nervous system tumours comprise 3.71% of the total tumours reported in males and 1.87% of those in females[35].

As many meningiomas are asymptomatic, autopsy study material show a higher incidence of meningiomas.

AGE – SPECIFIC INCIDENCE RATES

Increasing trends are seen with increasing age. From an incidence rate of 0.12 in the age range

of 0-19 year the rate increases to 18.86 after the age of 80 years.

The ICMR study[35] has shown the percentage of brain tumours by the 5 year age group to be 3.33% in males and 1.94% in females[35].

Incomplete reporting, inadequate follow up studies and potential selection biases have limited the estimates of mortality associated with meningiomas in the West and more so in India.

However overall survival rates are :

2 year survival rate : 81%

5 year survival rate : 69%

Population based survival studies from the 1990's in the United States have put the 5 year survival rate as ranging from 73% to 94%[2].

Table 1

AGE SPECIFIC INCIDENCE RATES

Claus et al[8]

Age	0 – 19	20 – 34	35 – 44	45 – 54	55 – 64	65 – 74	75 – 84	85+
Rate	0.12	0.74	2.62	4.89	7.89	12.79	17.04	18.86

SEX SPECIFIC INCIDENCE RATES

There is a pronounced female predominance, particularly with reference to the benign category of meningiomas[24].

MALE TO FEMALE RATIO

1 : 1.4 to 1: 2.8

Hormone receptor status appears to be the main reason behind this females predominance. But the incidence in childhood remain equal among boys and girls with some studies demonstrating a male predominance.

Furthermore, men are found to have a higher incidence of the more ominous atypical and anaplastic meningiomas.

RACE :

Meningiomas are more prevalent in Africa than the West., even there African – Americans form a major part of the population diagnosed with meningiomas[9].

RISK FACTORS ASSOCIATED WITH THE DEVELOPMENT OF MENINGIOMAS

The two factors for which the strongest evidence exists with respect to an association with meningioma risk are

1) Exposure to ionizing radiation

2) Hormones

Ionizing Radiation

Ionizing radiation increase the risk of intra cranial tumours particularly meningiomas, probably by damaging the DNA[9].

Mann et al reported the first case of radiation induced meningioma following high dose radiation to the orbit in a four year old child following the resection of an optic nerve glioma.

Commonly meningiomas occur after radiation therapy for pituitary adenoma, glial tumours and scalp abnormalities like tinea capitis.

Dental radiographs are another important causative factors of meningiomas. A population based case control study by the University of Washington Seattle shows that dental X-rays do cause meningiomas[26].

Meningiomas also occur following high dose radiation therapy for other primary intra cranial tumours.

Factors like tissue vulnerability, radiation type and dose, underlying disease and additional chemotherapy may influence tumorigenesis of meningiomas after another primary intracranial neoplasm has been irradiated.

Dore et al[26] found that the majority of secondary tumours were within the margin region of the treatment volume, when the volume received was less than 6 gy.

The average time of tumour induction is twenty one years after high dose therapy compared with thirty five years following low dose therapy.

A recent case control study of 200 meningioma reported on odds ratio of 2.06 (C.I : 95%) showing a strong association with radiation and meningiomas.

Hormones

Several factors point to the association of meningioma and hormones.

1) Increased incidence of the disease in women compared to men

2) The presence of progesterone, rarely estrogen and sometimes androgen receptors in meningiomas.

3) The association of breast cancer and meningiomas[13].

4) Change in the size of meningiomas during the luteal phase of the menstrual cycle and pregnancy.

Endogenous Hormones :-

Exposure to hormones may be exogenous or endogenous. A population based study of brain tumours from the late 1980s found that women with natural or surgical menopause (RR 0.59 – 0.12) had a reduced risk of meningiomas[9].

Similarly in the Nurses Health Study[48] the relative risk for meningiomas was less for candidates with late menopause. This study has also observed an increased risk for parous women compared with nor parous women. But other studies have found pregnancy protective.

Exogenous Hormones :-

Only recently have researchers started addressing the issue of exogenous hormones in the form of hormone replacement therapy and oral contraceptives. In a case control study inserted within the Nurses Cohort study[9] that included 125 meningiomas an overall positive association with hormone replacement therapy was found. Another case control study from three Chicago hospitals between 1987 and 1992 reports a protective effect for contraceptives[9].

A retrospective cohort study using the Mayo Clinic Jacksonville patients database confirms the

positive association of HRT and meningiomas[9].

These slightly contradictory findings emphasize the need for larger well controlled population based studies.

Head Trauma :

Since the time of Harvey Cushing, trauma has been suggested as a risk factor for meningiomas. Several small case studies from the 1980's report a positive association with meningioma.

In a cohort study of 228,055 Danish residents hospitalized for head trauma between 1978-1992 the standardized incidence ratio was 1.2 (95% CI) [9].

Cell Phone Use :

Exposure to the electromagnetic fields from cell phone use and the subsequent meningioma risk, though of great public interest has not been substantiated. This is partly because cell phone use began a relatively short while ago and the few conducted studies were of small sample size.

Breast Cancer Association With Meningioma

Common risk factors and shared genetic predisposition may be the reason why meningiomas are seen commonly associated with breast cancer. Studies have shown that BRCA1 or BRCA2 mutations are not found in sporadic meningiomas.

The relative risk for the association between breast cancer and meningioma observed across currently existing studies range between 1.5 and 2.0 with the majority being statistically significant.

Allergy :

Contrary to the positive association between glial tumours and allergy, no such association has been found for meningiomas. Few large population based studies have proven this non – association.

Viruses :

Though several viruses have been implicated, papovavirus DNA has been found in meningiomas. But a definite role for viruses is yet to be proven[9].

CYTOGENETICS :

Consistent cytogenetic abnormalities are found between 60 and 100% of meningiomas, depending on the various epidemiological factors and the laboratory technique used.

Both chromosomal loss and structural abnormalities are found. The most frequent chromosomal abnormality is simple monosomy of chromosome 22 in about 70-80% of cases. Less often a deletion of Ch. 22q occurs instead of simple monosomy. Of the other chromosomes Ch 14, is frequently involved in non-random loss or structural rearrangements[2]. Chromosomes 1, 7, 14 and Y and to a lesser degree 18, 19 and 20 are also noted.

A recent cytogenetic study has raised the possibility of chordoid meningiomas being associated with a characteristic translocation of chromosome arms 1p and 3p : t(1 : 3) (p12 – 13, q11).

In the early stages of development meningiomas are likely to have a normal karyotype or a simple monosomy. As the neoplasm progresses the karyotype becomes increasingly hypodiploid and later structural rearrangements may occur[13].

The increasing grades of the meningiomas are associated with chromosomal changes of increasing complexity.

In a study of 765 meningiomas the grade I meningiomas were found largely to have a normal karyotype, or monosomy 22. Grade II meningiomas were found to have both numerical and structural abnormalities. Fibroblastic meningiomas were found to have telomeric associations[9].

Meningiomas of the convexity were often grade II or III with loss of chromosome 22 and complex karyotypes.

The consistent losses of 22q followed by 1p, 1O and 14q strongly suggests the presence of meningioma tumour suppressor genes on these chromosomal arms. Analysis of polymorphic loci in DNA has supported the hypothesis that the loss of a tumour suppressor gene from the long arm of chromosome 22 to be the initiating genetic event in the pathogenesis of meningiomas[34].

NF – 2 gene in meningiomas :

Mutations in the NF2 gene are found in upto 60% of benign meningiomas and are probably inactivating mutations. Most of there mutations are small insertions or deletions or nonsense mutations.

Among benign meningiomas, the frequency of NF2 mutations varies. Both fibroblastic and transitional variant carry NF2 gene mutations in 70-80% of cases while meningiothelial meningiomas to so only in 25% of cases.

Chromosome 22q may harbour a second meningioma gene. β - adaptive gene, found in a region of 22q, has been found to be disrupted by translocations in several meningiomas.

Atypical and malignant meningiomas consistently show losses in 1P, 10 and 14q. Loss of chromosome 10 is associated with morphological signs of malignancy[2].

SPECIALISED CATEGORIES OF MENINGIOMAS

Radiation Induced Meningiomas

Rubenstein et al[34] defined radiation induced meningiomas as a separate nosological entity due to distinct clinico pathological featuers with regard to their presenting symptoms, multiplicity, recurrence rate and histopathology.

They characteristically occur in younger populations who have received a high dose of ionizing radiation. This suggests that the effect of radiation on the younger vulnerable meninges is more.

Clinically the hallmarks of radiation induced meningioma are alopecia and an atrophic scalp that overlies the meningioma.

The average time to tumours induction is 21 years for high doses and 35 years for low doses indicating that the chromosomal injury caused by higher doses elicit more rapid loss of cell control mechanisms and earlier expression of the neoplastic phenotype[34].

The frequency of atypical meningiomas is more among radiation induced cases. Similarly multiplicity is far more common. But surprisingly proliferation indicies like chromo deoxy uridine labeling indices, PCNA and Ki – 67 labelling indices show a slow growth potential[2].

Cohan et al have laid down the following criteria to label a meningioma as “radiation – induced”

1. The tumour must occur within the field of irradiation.
2. The tumour must differ from any pre existing neoplasm.
3. It must occur after a reasonable interval, sufficient to demonstrate that the neoplasm did not exist prior to radiation.
4. Significantly higher incidence in the irradiated groups compared to the control groups.

Studies have long stressed the need for life long clinical and radiological surveillance of cranially irradiated patients.

ATYPICAL AND ANAPLASTIC MENINGIOMAS :

Various sources put the incidence of meningiomas at 13-26% of all intracranial tumours. A minority of these tumours possess aggressive qualities. Atypical meningiomas constitute about 4.7 – 7.2% of all meningiomas while anaplastic meningiomas comprise 1-2.8%[2].

2% of all benign meningiomas transform into more malignant forms, while 28.5% of all recurrent benign meningiomas turn out to be atypical or anaplastic[30].

They are more common in men than women.

Additional genetic mutations are responsible for atypical and anaplastic features.

Atypical meningiomas carry mutations of Ch 22q, a gain of 1q, 9q, 15q and 17q or lss of 1p, 6q, 10q, 14q or 18q.

Anaplastic meningiomas are fund to have further mutations like amplification of 17q and loss of 9p.

CLINICAL PRESENTATION OF MENINGIOMAS :

Meningiomas produce symptoms by several mechanisms like irritation of the underlying cortex, compression of the brain or nerves, hyperostosis of the overlying bone, soft tissue invasion and vascular compromise[10]. Hyperostosis does not always occur due to direct bony permeation but may be a consequence of increased sub-periosteal bone formation stimulated by impaired blood supply. Meningiomas contain high levels of alkaline phosphatase that is known to possess indirect ossifying

properties.

Characteristic symptoms are produced by site specific meningiomas.

Para Sagittal	:	Monoparesis of contralateral leg
Sub-frontal	:	Change in mentation
Olfactory groove	:	Foster – Kennedy syndrome
Cavernous sinus	:	Multiple cranial nerve deficits
Occipital lobe	:	Contralateral hemianopia
Cerebello-pontine angle	:	Decreased hearing
Spinal cord	:	Back pain, Brown – Sequard syndrome, paraparesis.

QUALITY OF LIFE :

Though classified clinically and histopathologically as benign, the clinical effects of meningiomas can be devastating. Upto 30% of patients operated for meningiomas cannot read, write, drive or even think at the same level as before their diagnosis.

DIAGNOSIS OF MENINGIOMA

No specific laboratory test are available for screening meningiomas. Levels of linolineic acid may raised in the serum[29]. Imaging studies are the mainstay of diagnosis.

X – RAYS :

Meningiomas may exhibit the following features on x- rays

- Hyperostosis of the overlying bone

- Increased vascular markings

- Intra-cranial calcifications

CT SCANS :

Dural based lesions that are iso-attenuating to hyperattenuating. They enhance homogenously and intensely after the injection of iodiated contrast material. Perilesional edema is usually marked. The underlying brain may be compressed. Multiple lesions are difficult to differentiate from metastasis.

MRI – MAGNETIC RESONANCE IMAGING :

On T1 and T2 weighted MRI's the tumours have variable signal intensity. Enhanced MRI's are imperative for a diagnosis of meningioma. Meningiomas enhance intensely and homogenously after injection of gadolinium gadopentate. Peri lesional edema may be more apparent on MRI than CT scans. An enhancing tail involving the duramater may be apparent on MRI.

ANGIOGRAPHY :

Angiography features of meningiomas demonstrate the following :

- Vascular supply from the external circulation

- So called " mother in law blush" a vascular blush that comes on early and leaves late.

- Sun burst or radial appearance of the feeding arteries.

PROGNOSTIC FACTORS IN MENINGIOMAS

The 2000 WHO classification of meningiomas provides a grading system for menigiomas based on histological criteria that has a pronounced impact on prognosis.

Over the years several authours like Rubenstein, Tallkalnin and Mahmood have attempted to classify and grade meningiomas but had inconsistent results.

Two well structured studes by Perry et al from the Mayo clinics have a simply worked out and very reproducible grading system[2].

The Mayo Clinic Grading Scheme Pathological Criteria

Atypical Meningioma

≥ 4 Mitosis / 10hpf

Or at least three of the following features

Sheeting

Macronucleoli

Small cell formation

Hyper cellularity (> 53 nuclei/hpf)

Brain invasion

Anaplastic meningiomas

≥ 20 mitosis / 10 hpf

or

Focal or diffuse loss of meningothelial differentiation resulting in a carcinoma, sarcoma or melanoma

like appearance.

HORMONE RECEPTOR STATUS

Some hormones and their receptors have been studied extensively in meningomas due to the increased growth rates of meningiomas with raised sese hormone levels.

Estrogen Receptors :

Donnell et al[1] first described the presence of estrogen receptors in four out of six meningiomas. They are fund at in a much lower percentage of cases when compared to progesterone receptors. Ligand binding and enzyme immunoassay does not reveal estrogen receptors but hand shift assays have found an estrogen like protein. Estrogen receptor related small heat shoed protein (HSP – 27) has also been demonstrated.

Data from the Brigham and Women's Hospital[1] has shown variations among the estrogen receptors iso-forms. 68% of cases express receptors alpha mRNA and 32% express beta mRNA. Both types are capable of binding estrogen and activating genes, but elicit different responses in different organs. A more detailed sub-typing of estrogen receptors may explain why anti-estrogen drugs like tamoxifen has yielded inconclusive results.

Progesterone Receptors

Progesterone receptors are found in 40-100% of meningiomas.

Hsu et al[47] have shown that benign menigiomas were more likely to be PR positive.

PR status is immensely related to the mitotic index and grade and therefore associated with a between prognosis.

Jay et al have shown that hormonal manipulation may modify the growth in some tumours.

In in vitro growth of meningioma cells, Koper et al suggested that the presence of progesterone in the culture medium increases the sensitivity of the meningioma cells to mitogenic stimuli like epidermal growth factor. Mifepristone a progesterone receptor blocking agent can counteract the effects of progesterone.

Studies by Grunberg et al have shown that mifepristone may play a role in tumour size reduction particularly in benign meningiomas.

High progesterone receptors expression are found in the following groups :

1. Male patients in the age group above 50 years.
2. Grade I meningiothelial meningiomas when compared to fibrous or transitional variants.
3. Meningiomas with a low MIB – 1
4. Atypical and anaplastic meningiomas show low progesterone receptor indices.

A recent paper by the American Cancer Society further studied the status f progesterone receptors and MIB – 1 status with relation to age. They have concluded that either index has no definite relationship with age and that atypical a anaplastic variants could occur in old age.

MIB – 1 LABELLING INDICES :

Predicting the biological behaviors and propensity for recurrence has roved to be notoriously difficult in meningiomas. various proliferation indices like MIB – 1 labelling indices have been tried.

MIB – 1 is a monoclonal antibody that detects Ki 67 antigen, a non-histone protein expressed only in the proliferative phase of the cell cycle i.e., during G1, S, G2 and (Mitotic) M phase. It was earlier considered to be a reliable marker for higher grade tumours. But larger scale studies have shown that a high MIB-1 index need not predict recurrence.

Other factors like tumour location, grade of excision of surgery and tumour histology are more predictive indications of recurrence.

SURGICAL GRADE OF EXCISION

Simpson's grading schemes[29] have categorized the extend of surgical resection for meningiomas.

Table 2

Grade I	Total Macroscopic Resection With Dural Attachment / Bone Involved Sinus
Grade II	Total resection and diathermy of attachment
Grade III	Total macroscopic resection of tumour only

Grade I resection had the best rate of recurrence when compared to grade III, emphasizing the need for complete resection.

SITE OF OCCURRENCE OF THE MENINGIOMAS

The highest rate of recurrence has been documented for olfactory groome meningiomas and basal meningiomas at 41.7% followed by cerebellopontine angle tumours and posterior fossa tumours. Recurrences are the least among convexity meningiomas.

MOLECULAR BIOLOGY OF MENINGIOMAS

Oncogene expression analysis has revealed a host of growth factor and their receptors in meningiomas.

Platelet derived growth factors (PDGF – A, PDGF – B and PDGF - β receptor) are found to be increased and they further raise C – fos levels. PDGF – A and PDGF - β receptors are involved in the growth control of meningiomas through autocrine and paracrine mechanisms.

In in vitro studies have revealed other powerful mitogenic agent like epidermal growth factor in meningiomas. Somatostatin receptors are present in all those tumours that contain EGF leading to interaction of the two.

Meningiomas also expressed insulin like growth factor 1 and 2 in excess of the amounts found in normal brain and meninges.

Suramin, a polyanionic compound that interferes with growth factor to cell binding has been shown to inhibit meningioma growth and holds therapeutic promise.

bFGF (basic fibroblastic growth factor) levels are increased and function as mitogens, angiogenic agents and differentiators. Intracellular calcium levels appear to play a key role in their voltage dependent signal transduction methods.

VEGF (Vascular endothelial growth factor) is a potent angiogenic agent that also acts as a vascular permeability molecule. The distinctive peri-tumoural edema is attributed to the effect of

VEGF.

Endothelin, a potent vasoconstrictor also acts as a growth promoting agent in meningiomas.

Down stream mediators of cellular proliferation like the multiple JAK/STAT molecules are particularly elevated in transitional meningiomas.

All these growth factors are being investigated as potential targets of molecular targeted therapy.

MATERIALS AND METHODS

This is a single Institution based study of all the meningiomas received at the Department of Neuropathology, Madras Medical College, during the period from January 2001 to August 2006.

The total number of central nervous system tumours received during this period were 1946, of which 342 were meningiomas.

All these 342 cases were diagnosed pre – operatively as meningiomas by imaging studies, either CT scans or MRI.

Medical records of all the patients were reviewed and supplemental clinical information was obtained from the treating physician where ever necessary.

The recorded clinical data include the patients age, sex, history of any significant co-morbidity, imaging findings, location of the tumour, date of surgery, extent of surgical resection and additional therapy given if any.

All the specimens were received and fixed in 10% buffered formalin and were manually processed.

Gross features like the overall size, shape, colour, consistency, cystic and necrotic changes, attached duramater and bone were evaluated. Where ever possible the specimens were bisected along the longitudinal diameter and a minimum of four bits each measuring 3 – 5 mm in thickness were taken. After manual processing sections of 3 – 5 micron thickness were cut and stained with hematoxylin and eosin. In some cases additional special stains like periodic acid Schiff and Verhoeff's Van Gieson were performed.

The meningiomas thus diagnosed were subtyped into various histological types.

Grading of the meningiomas was done as per the WHO 2000 classification of Central nervous system tumours into grade I, II and III.

The distribution of these various histological types of meningiomas with reference to age, sex and site of occurrence were analysed and compared with data from other studies.

Further, recent literature regarding the epidemiology, clinical presentation, etiopathogenesis, grading and other prognostic factors were reviewed.

PROCEDURE FOR HEMATOXYLIN AND EOSIN STAINING

1. Dewax the section, dehydrate through graded alcohols and bring sections to water.
2. Remove fixation pigments if necessary, stain in haematoxylin for 5 minutes.
3. Wash well in running tap water.
4. Differentiate in 1% acid alcohol for 2- 4 seconds.
5. Wash well in running tap water until sections are blue again for 15 – 20 minutes.
6. Stain in eosin for one minute
7. Wash in water for 5 minutes
8. Dry the sections, clear in xylene, mount with DPX, and label the slide

OBSERVATION AND DISCUSSION

At the Department of Neuropathology, Madras Medical College, a total of 2702 specimens were received for histopathological examination during the period extending from January 2001 to August 2006.

Out of these 2702 specimens there were 1946 central nervous system neoplasms, which encompassed a wide range of lesions including gliomas, meningiomas, oligodendrogliomas, schwannomas, etc.,

Among them gliomas formed the largest category with a total of 623 cases.

Our study showed that the total number of meningiomas received during this five year period was 342. They formed the second largest category after gliomas. Other tumour of the meninges too were reported during this period, which include 9 haemangiopericytomas. These tumours were not taken up for this study as they have been excluded form the category of meningiomas by the new WHO 2000 classification.

As per the WHO 2000 classification there were 320 grade I tumours, 11 grade II tumours and 11 grade II tumours.

AGE SPECIFIC INCIDENCE :

In our study there was a wide age range with the youngest candidate being 5 years of age and the oldest 78 years of age.

The peak incidence was found in the fifth decade with 104 cases contributing to 30.40% of the cases. The least incidence was recorded in the eight decade with only a single 78 year old man in the entire 5 year study period.

The number of cases in the first decade too was low with 10 cases in 5 years.

The frequency of meningiomas at our institute as per our study is 17.47% (342 meningiomas out of 1946 CNS neoplasms).

This finding is comparable with statistics quoted in literature[9] that places the frequency of meningiomas at 20%.

According to Indian literature meningiomas account for the second largest group after gliomas. The statistics from our study are comparable with that of a similar hospital based study of meningiomas conducted at Bombay Hospital by A.B. Shah et al[42]. They too found that meningiomas were the second largest group with 267 meningiomas out of a total of 1321 CNS space occupying lesions.

INCIDENCE OF MENINGIOMAS OVER THE STUDY PERIOD

The incidence of meningiomas at our Institute over these five and half years has not been constant but subject to fluctuations.

Table 3

DISTRIBUTION OF CASES OVER A FIVE AND HALF YEAR PERIOD

Year	No. of cases
2001	58
2002	43
2003	54
2004	69
2005	48
2006 (upto August)	65

The highest incidence has been the year 2006 with 65 cases being reported over a period of eight months. The average number of meningiomas reported per year is about 62 at our Institute

DISTRIBUTION OF MENINGIOMAS OVER THE STUDY PERIOD

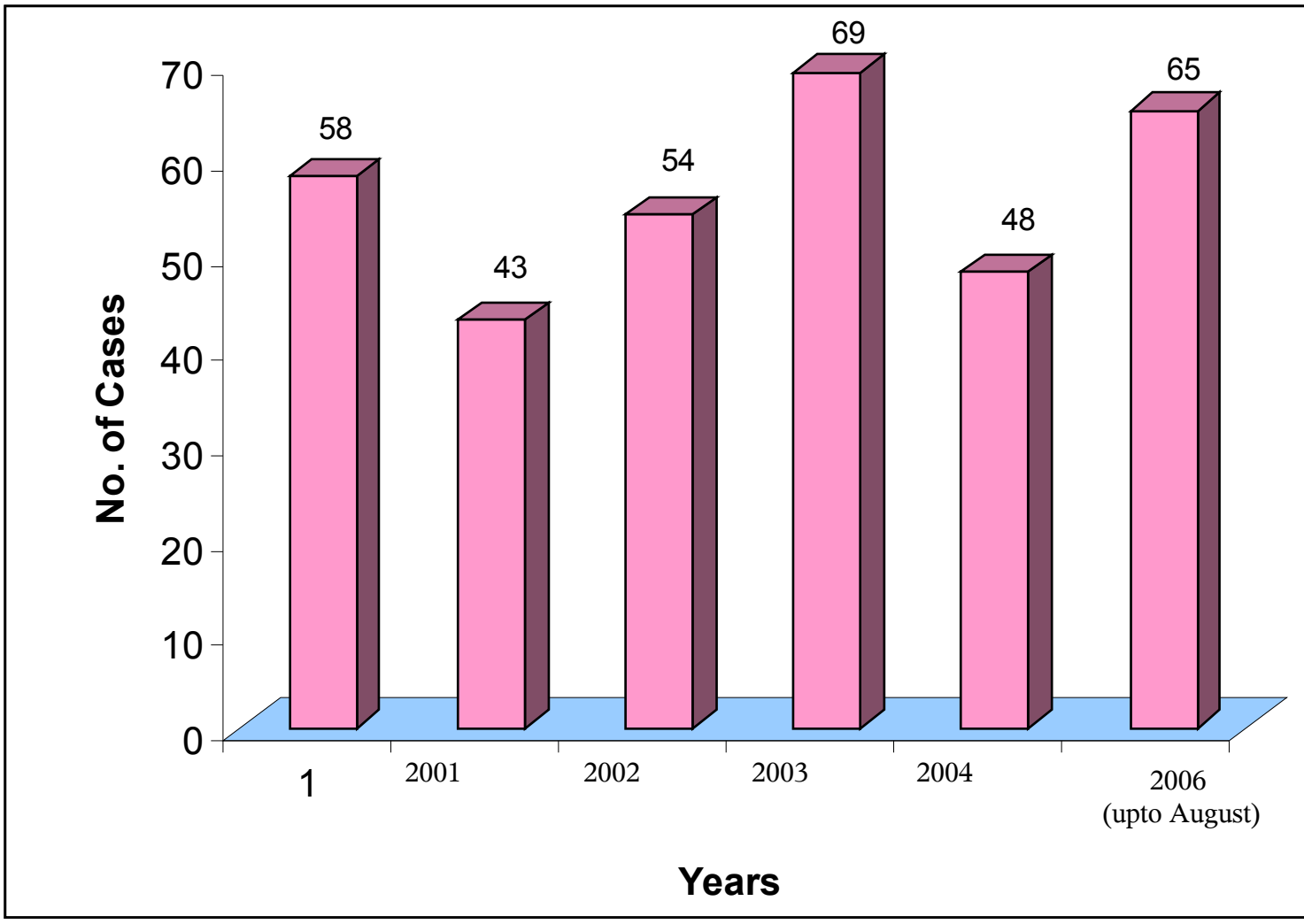


Table No. 4

AGE DISTRIBUTION OF MENINGIOMAS

Age group (Years)	No. of cases n = 342	Percentage
0 – 10	10	2.92%
11 – 20	18	5.26%
21 – 30	48	14.03%
31 – 40	91	26.60%
41 – 50	104	30.40%
51 – 60	47	13.74%

61 – 70	23	6.72%
71 – 80	1	0.29%

Our study showed a gradually increasing age specific incidence rate from the first decade onwards. This peaked at the fifth decade with 104 cases forming 30.40%.

The incidence rate thereafter tapered off till the eight decade which showed only a single reported case.

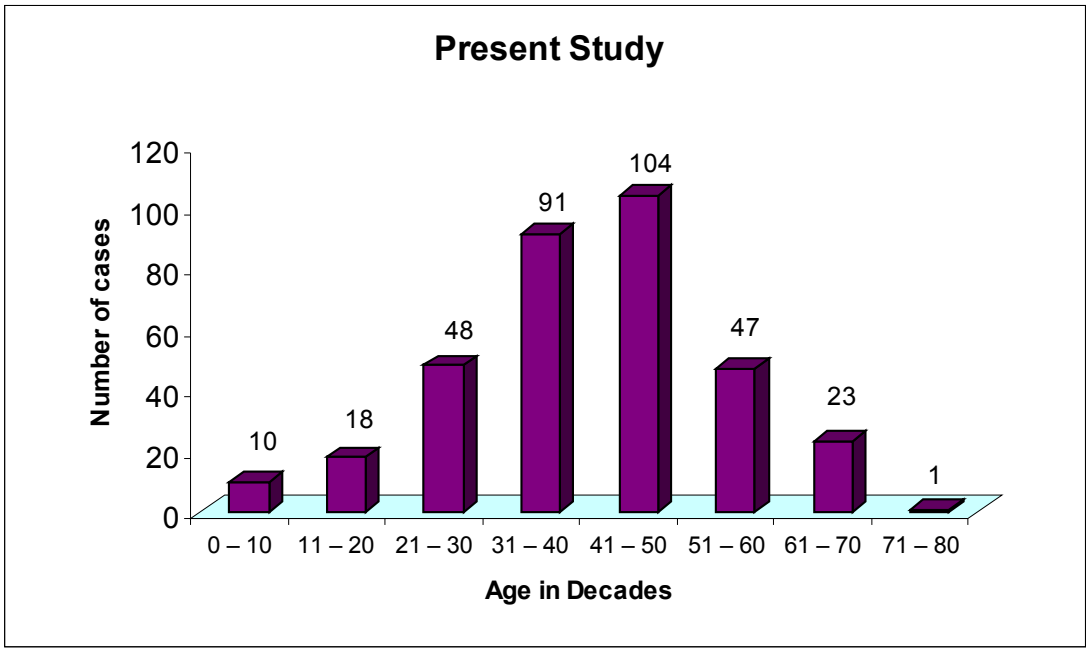
Literature from the west[9] shows that the age specific incidence rate rises from childhood onwards and reaches a peak in the 8th decade. The findings in our study and in the study at Bombay Hospital do not corroborate with the same. A.B. Shah et al[42] too showed a peak incidence in the fifth decade with 26% of their cases falling in this age group.

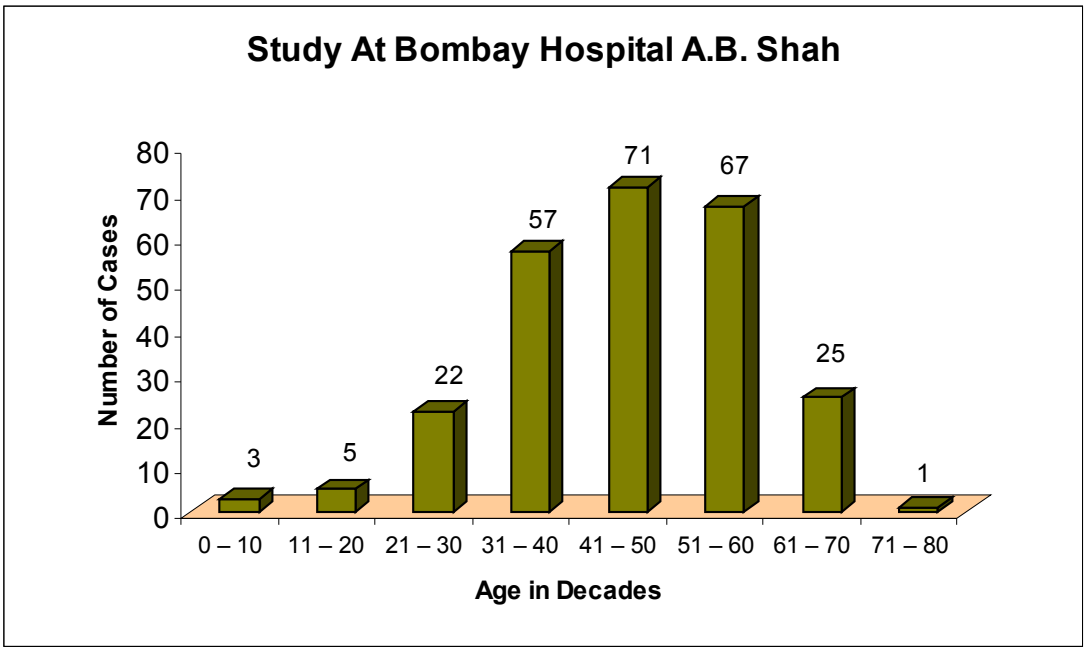
The reasons why in India statistics tend to remain low in the elderly age groups are probably poor availability of health services to the aged when compared to their western counterparts.

AGE DISTRIBUTION OF MENINGIOMAS

Most of the patients in our hospital hail from poor socio – economic groups, particularly from rural areas. Several elderly patients have associated co – morbid conditions like diabetes mellitus, hypertension and ischemic heart disease, which increase the risk for anaesthesia. Further many elderly patients and their families show a general reluctance to undergo a major neurosurgical procedures with all its attendant risks. All these factors along with a general lack of awareness of the condition may be the reason for the low incidence of meningiomas in the older age groups in India.

Meningioma Age Distribution



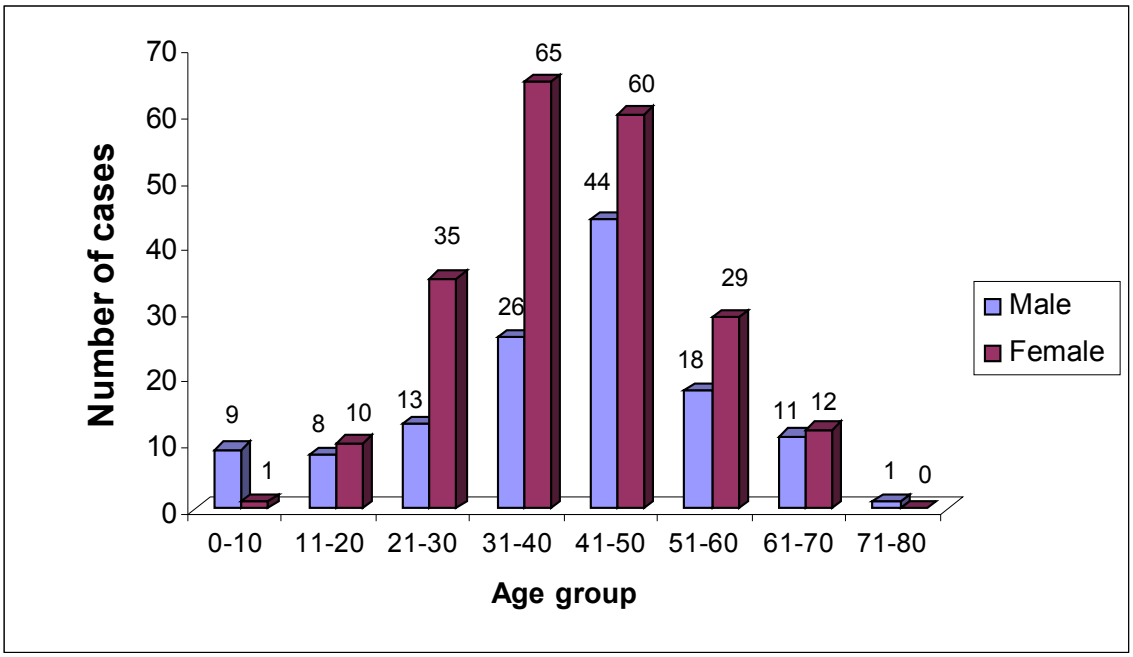


SEX – SPECIFIC INCIDENCE RATES

In our study among a total number of 342 meningiomas, there were 212 female patients and 130 male patients. The sex specific incidence rate for women was thus 61.98% and that for men was 38.01%.

This overall female predominance was not seen in the pediatric age group. In the first decade, out of a total of 10 cases there was a pronounced male predominance with 9 males and one female. There findings correlate with other studies on childhood meningiomas as in the one by E.J. Rushing et al[10]. Of 87 child hood meningiomas studied by them, there were 52 males and 35 females.

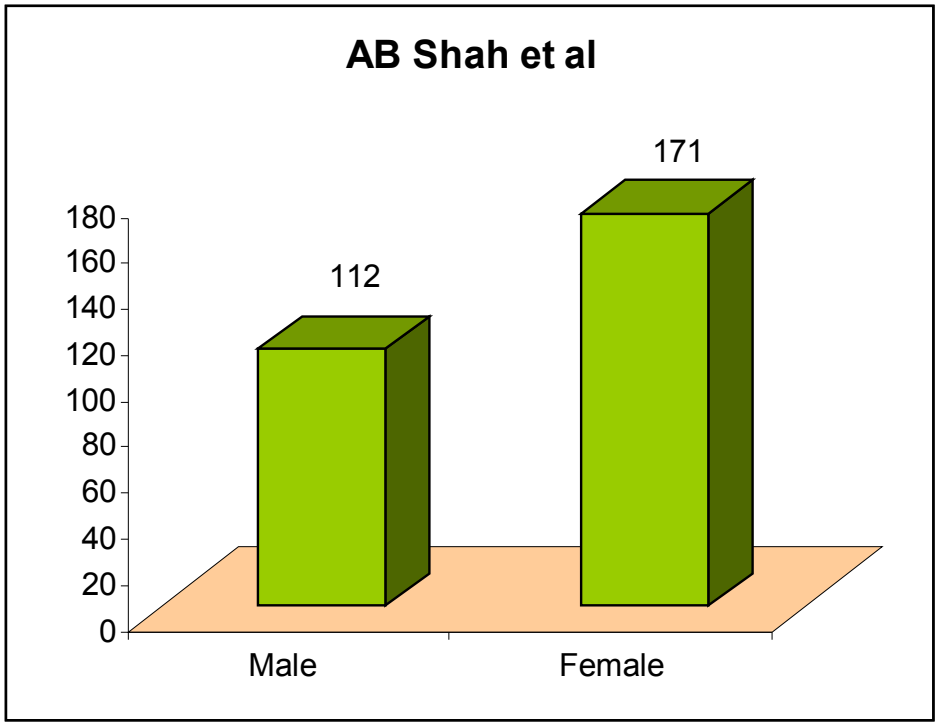
AGE AND SEX DISTRIBUTION OF MENINGIOMAS

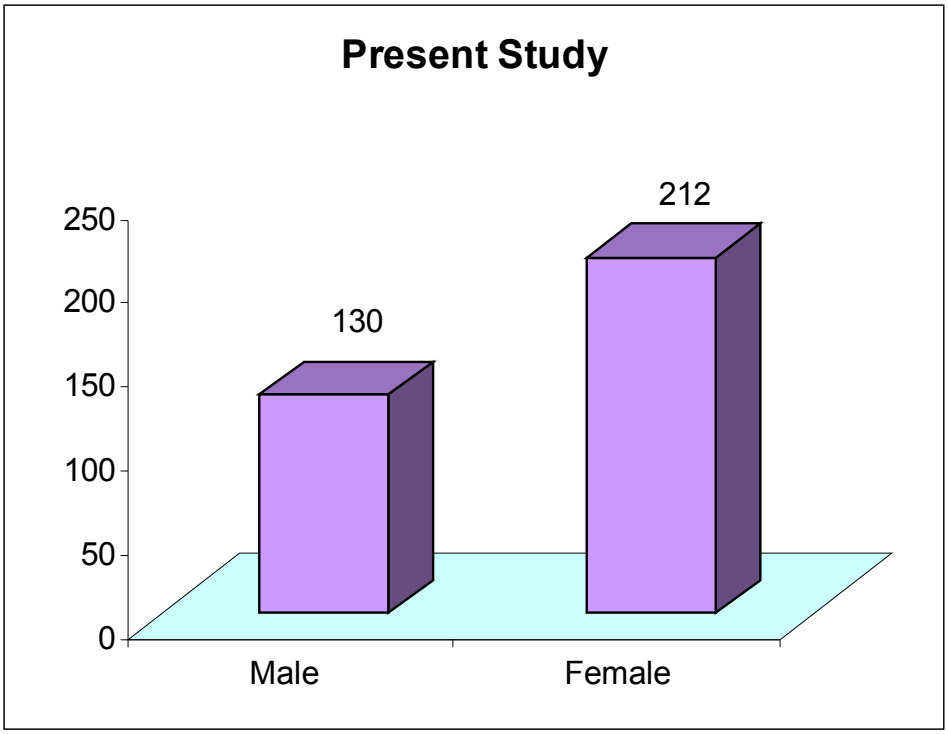


A study by Claus et al on the epidemiology of meningiomas show that women have a higher incidence of meningiomas[9]. The overall incidence rate is reported to be 38% for women compared to 20% in men.

Our study showed the largest number of female patients in the fourth decade with a total of 65 women. This predominance of women can be partly explained by the fact that meningiomas are hormone dependent. Progesterone specially acts as a potent growth factor[1]. Our study had two women who were pregnant when diagnosed with meningioma. Surgery was performed in the post partum period in both these women. Both women experienced an increase in the size of the tumour during pregnancy.

SEX DISTRIBUTION OF MENINGIOMAS





CLINICAL FEATURES :-

Our study revealed that the symptoms and signs among various patients varied according to the site of the tumour.

The most common presenting symptoms were headache associated with vomiting or nausea. 52% of all the cases had these symptoms. New onset seizures were the second most common symptoms accounting for 20% of all the cases. Children in particular presented with seizures more often than any other symptoms.

Other commonly observed symptoms were urinary incontinence, memory disturbances, behavioural abnormalities twitching of the face, derivation of mouth, rarely hemiparesis or hemiplegia. These symptoms were observed mostly in supra – tentorial meningiomas. Some patients experienced drowsiness or were even admitted in a comatose condition.

Orbital meningiomas presented with progressive proptosis, reduction of visual acuity and rarely with total blindness.

Spinal cord meningiomas presented with back pain in 80% of the cases. Some patients presented with paraparesis, numbness in the lower limbs and difficulty in getting up or walking. Two patients with spinal cord lesions in the 6^{th} decade presented with pathological features.

Cerebellopontine angle meningiomas manifested with ataxia, nystagmus, or reduction in hearing.

Three patients presented primarily with bony swellings of the scalp which later were diagnosed as underlying meningiomas with osseous involvement.

Exacerbation of symptoms during the course of pregnancy were noted in both women who were

diagnosed during their pregnancy.

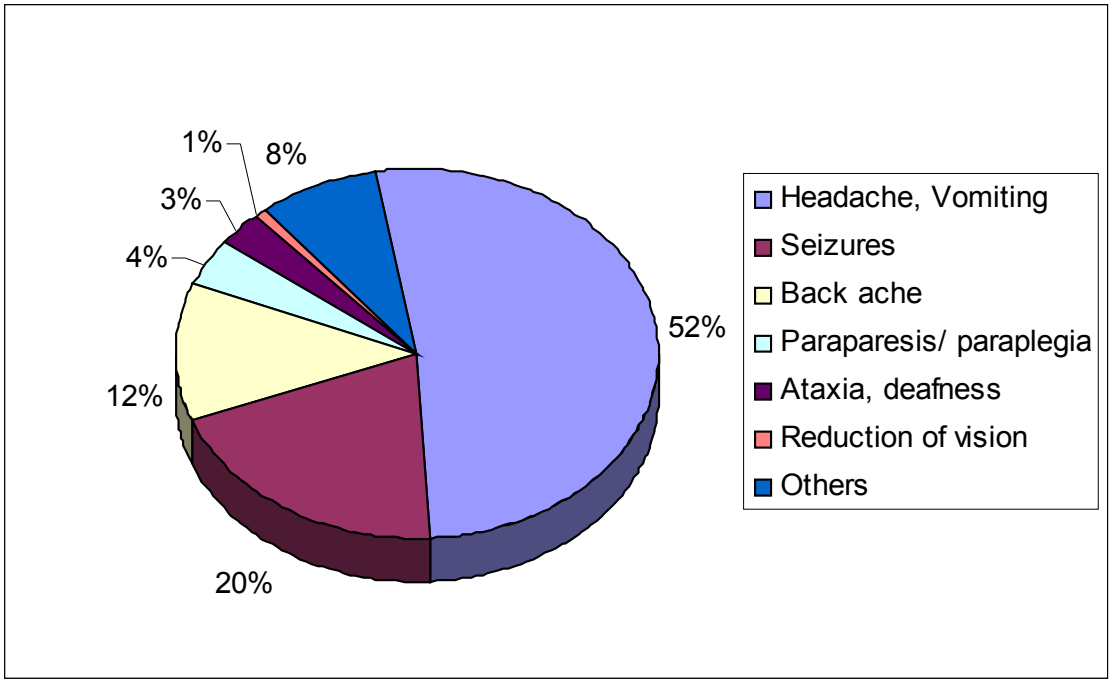
In our study four patients had a history of previous head trauma. Three male patients developed meningiomas at the site of head injury following road traffic accidents after a mean period of five years. One female patient reportedly developed a meningiomas 3 year after a fall from a height.

There were two patients who had a history of cranial surgery but no case of cranial irradiation was noted in our study.

Table 5 :- CLINICAL FEATURES IN MENINGIOMAS

Signs	Frequency (n =342)
Headache, Vomiting / Nausea	52%
Seizures	20%
Back ache	12%
Paraparesis / paraplegia	4%
Ataxia, deafness	3%
Reduction of vision	1%
Others	8%

CLINICAL FEATURES OF MENINGIOMAS



CO – MORBID CONDITIONS

Several elderly patients in our study had co – existing morbid conditions like diabetes mellitus, ischemic heart disease and hypertension.

Some patients had so – existent tumours while some were treated earlier for other malignancies. One 12 year old boy had a history of a schwannoma operated about a year before diagnosis of a spinal cord meningioma.

A 37 year old female had a co – existent neurofibroma, with a meningiothelial meningioma. Both were operated upon in two different surgeries.

Two women, both in the fifth decade were known cases of carcinoma cervix: one stage III, the other stage II. Both had undergone surgery and radiotherapy on an average of five years before being diagnosed with meningiomas.

A forty year old male with a left sided sphenoid wing angiomatous meningioma had Foster –

Kennedy syndrome.

Literature states that meningiomas occur commonly in patient with neurofibromatosis type (NF2), Gorlins' syndrome and Down's syndrome[10]. Several co – existent tumours have been reported along with meningiomas like medulloblastomas, primitive neuroectodermal tumours, schwannomas, astrocytomas, retinoblastomas and rarely cases of leukemia.

The three cases of lympho – plasmacytic meningiomas reported in our study did not have monoclonal gammopathy or endocrine abnormalities but two of then had anaemia.

Similarly chordoid meningiomas are closely associated with Castleman's disease but the single reported case in our study did not[4].

The association of meningiomas with breast carcinomas too is well known. We did not see any case with a history of breast carcinoma[1]. Follow up studies were inadequate as most cases were lost to follow up.

IMAGING STUDIES

Out of the total 342 meningiomas in our study imaging study details were available for 237 cases. Of these computerized tomography (CT) scans were done pre – operatively in 181 cases while magnetic resonance imaging (MRI) scans were done in 56 cases.

Analysis of the results of computerized tomographic imaging of meningiomas revealed that hyperdense lesions numbered 96 out of the 181 cases done. Most of these cases were fibrous or meningiothelial meningiomas. No site or age predilection was noted. Isodense lesion on CT scans numbered 46 while hypodense lesions were 20 in number. Mixed dense lesions were 19 which included two papillary, one rhabdoid and four atypical meningiomas.

Most of the lesions showed extensive perilesional edema.

In those cases where contrast enhanced CT scans were done, brilliant enhancement with contrast was noted. Intra tumoural calcification was picked up, in many psammomatous meningiomas.

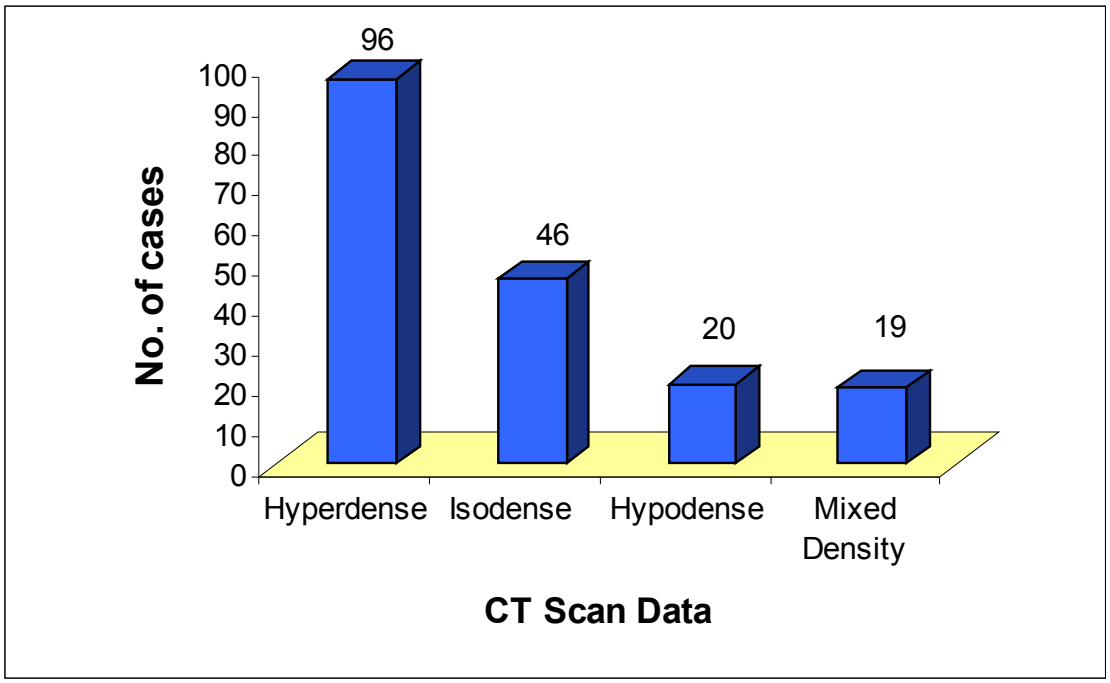
Magnetic resonance imaging showed that 35 cases were hypointense on T1 weighted MRI and hyperdense on T2 weighted MRI, while 21 cases were iso intense on T1 weighted MRI hyperdense images on T2 weighted MRI which 21 cases were iso intense on T1 weighted MRI hyperdense images on T2 weighted MRI.

Perilesional edema was appreciated better on MRI scans than CT scan studies.

Table No.6 : CT SCAN DATA DISTRIBUTION

Image findings	No. of cases n = 181	Percentage
Hyperdense lesions	96	53.03%
Isodense Lesions	46	25.41%
Hypodense Lesions	20	11.01%
Mixed Dense Lesions	19	10.49%

CT SCAN DATA DISTRIBUTION



SITE SPECIFIC INCIDENCE OF MENINGIOMA

Of the total number of 342 meningiomas, the vast majority of them were intracranial in location numbering 303. The remaining 39 meningiomas were intraspinal in location.

Among the intracranial tumours most were supratentorial and dural based.

The most common location for the intra cranial tumours were the frontal lobes followed by the parietal lobes.

The distribution of the studied meningiomas among various sites is given below.

Table 7

Location of the tumour	No. of cases n = 342	Percentage
Frontal	63	18.42%
Parietal	62	18.12%
Para Sagittal	13	3.80%
CP Angle	20	5.84%
Sphenoid wing	23	6.72%
Posterior Cranial Fossa	14	4.09%
Falx	24	7.01%
Temporal	20	5.84%
Suprasellar	8	2.33%
Intraventricular	10	2.92%
Orbital	6	1.75%
Clival	1	0.29%
Basal	2	0.58%
Spinal Cord	39	11.40%
Miscellaneous	47	13.74%

SITE WISE DISTRIBUTION OF MENINGIOMAS

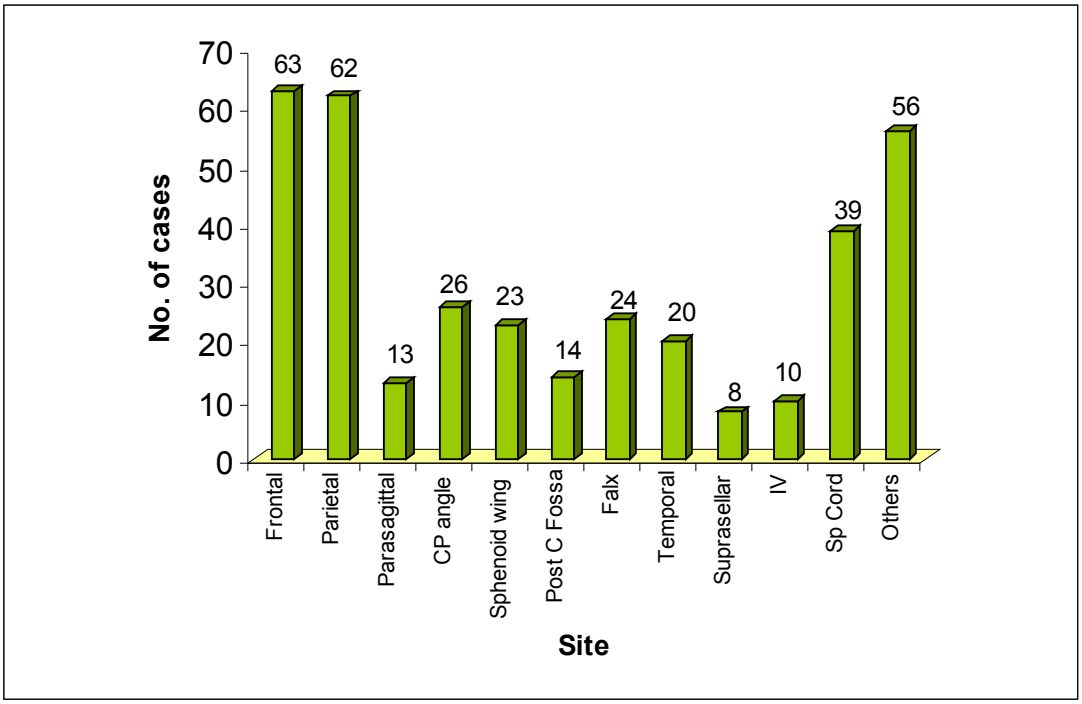


Table 8

COMPARISON OF ANATOMIC LOCATIONS OF INTRACRANIAL MENINGIOMAS

Location of the tumour	PRESENT STUDY		A.B. SHAH et al	
	No. of cases n = 342	Percentage	No. of cases n = 247	Percentage
Frontal	63	18.42%	69	28%
Parietal	62	18.12%	33	13%
Middle cranial fossa	59	17.25%	36	15%
CP Angle	20	5.84%	24	9%
Suprasellar	8	2.33%	17	6%
Temporal	20	5.84%	16	6%
Falx	24	7.01%	12	5%
Posterior Cranial Fossa	14	4.09%	16	6%
Miscellaneous	90	26.31%	24	9%

These figures corroborate with other studies. The epidemiological study by A.B. Shah et al[42] from Bombay Hospital show similar statistics.

Certain site specific meningiomas carry prognostic and surgical implications.

INTRAVENTRICULAR MENINGIOMAS

Intraventricular meningiomas are considered rare tumours. The origin of these tumours can be traced to embryological invagination of arachnoidal cells into the choroid plexus. Our study revealed ten meningiomas located intraventricularly. Bhatoe et al[3] reported 12 cases, from the Army Hospital, New Delhi. All were slow growing tumours and most of them were angiomatous meningiomas.

Out of 10 cases, 8 tumours were located in the lateral ventricles, one tumour in the third ventricle and one in the fourth ventricle. There were six meningothelial meningiomas, two fibrous meningiomas, one angiomatous meningioma and one papillary meningioma. They were no different histologically from dural based examples.

Sphenoid Wing Meningiomas

Sphenoid wing meningiomas are considered challenging to the neurosurgeon due to involvement of the adjacent bone[31]. The site of tumour origin, and the presence or absence of the arachnoidal plane between the tumour and cerebral vessels affect resectability.

Though most are benign meningiomas they occupy the parasellar region with its complex anatomical boundary zone between orbital and intracranial compartments including the cavernous sinus, prejudicing radical surgery.

Sphenoid wing meningiomas with osseous involvement are considered a separate entity by most neurosurgeons. Grossly these meningiomas may be of two types : a globoid variant and an en plaque

variant[11]. The latter form is characterized more by its clinical and radiological appearance rather than histology. They are more likely to produce hyperostosis or even show direct infiltration of the sphenoid wing.

Hyperostosis due to meningiomas :

The cause of associated hyperostosis in meningiomas at the sphenoid bone remains a point of controversy – specifically regarding whether this represents a secondary change of the bone without tumour invasion or a direct infiltration of the bone by the tumour.

The mechanism by which meningiomas accomplish this extensive invasion of bone may be due to preceding trauma, vascular disturbances enzymatic reactions, or stimulation.

Impaired blood supply due to tumour growth may induce increased sub periosteal bone formation. The level of alkaline phosphatase which is known to possess indirect ossifying properties is found to be three times higher in meningiomas with osseous involvement than those without[5].

Pompili et al[11] studied and demonstrated that there is true hyperostosis with formation of additional bone and that the invasion of bone is not merely a lytic process.

In spite of this seemingly aggressive growth pattern, sphenoid wing meningiomas are not different histologically from those occurring at other sites. In our study there were 23 sphenoid wing meningiomas.

There were 12 meningothelial meningiomas making up the largest category. There were four fibroblastic meningiomas, three angiomatous meningiomas, two transitional meningiomas, one metaplastic variant and one atypical example.

Table 9 shows the comparison between the study by F.Roser et al and the present study.

DISTRIBUTION OF HISTOLOGICAL TYPES AMONG SPHENOID WING MENINGIOMAS

Histological Type	Study by F.Roser et al n = 82		Present study n= 23	
Meningiomas	No. of cases	Percentage	No. of cases	Percentage
Meningothelial	58	70.73%	12	52.17%
Fibrous	4	4.87%	4	17.39%
Transitional	5	6.09%	2	8.69%
Atypical	5	6.09%	1	4.34%
Others	10	12.19%	4	17.39%

We found a slightly higher incidence of fibrous and angiomatous meningiomas and a lower incidence of atypical examples.

SPINAL CORD MENINGIOMAS

Our study showed extra cranial meningiomas located only in the spinal cord. Other sites reported in the literature include lung, ovary, soft tissue etc., but we did not encounter any such examples.

Spinal cord meningiomas occurred across all age groups and occurred in both sexes with equal frequency.

The most common site of occurrence was the thoracic spine with a predilection for the 6^{th} – 8^{th} thoracic vertebrae.

Of the studied 39 spinal cord meningiomas, psammomatous meningiomas were the

predominant type with 23 cases making up 58.97%. Meningiothelial meningiomas were the second most common category with 8 cases.

A study by Walter Reed Army Medical Centre, spinal cord meningiomas demonstrated a predominance of psammomatous types too. Childhood meningiomas presenting in the spinal cord too tend to be psammomatous in types.

Table No.10

HISTOLOGICAL VARIANTS OF SPINAL CORD MENINGIOMAS

Types	No. of Cases n = 39	Percentage
Psammomatous	23	58.97%
Meningiothelial	8	20.51%
Fibrous	4	10.25%
Transitional	3	7.69%
Angiomatous	1	2.56%

MULTIPLICITY IN MENINGIOMAS

Multiple synchronous tumours were encountered in three patients in our series. A thirty year old female with recent onset seizures and loss of consciousness was diagnosed to have multiple meningiomas by MRI scan. They were located in the corpus callosum, (R) parietal lobe, (R) occipital

lobe and (L) para – sagittal area. All four tumours showed features of meningiothelial meningioma. A 27 year old male presented with headache and vomiting of short duration. CT scan findings showed one tumour in the (L) fronto parietal area and one in the (L) sphenoid bone. Histopathological examination of the frontoparietal mass showed a fibrous meningiomas while the (L) sphenoid meningiomas turned out to be a psammomatous variant.

An elderly male patient aged 66 years presented with diminution of vision. CT scans showed bilateral occipital tumours, that were found to be anatomical distinct from each other during surgery. Both tumour were meningothelial meningiomas.

Multiple meningiomas are usually reported to occur in patient with neurofibromatosis 2 (NF – 2) [24]. Though there are various criteria for the diagnosis of NF – 2, all require either a family history of NF – 2 or presence of a vestibular schwannoma. All three of our patients with multiple meningiomas had neither. Families with multiple meningiomas have been reported without chromosomal 22q reported without chromosomal 22q deletions and it is hypothesized that this disorder may result from alterations in other negative growth regulators important for meningial cell growth and differentiation.

None of our reported patients had other features of neurofibromatosis like café – au – lait spots, subcutaneous neurofibromas, axillary freckling, Lisch nodules or bony dysplasias.

Aggrawal et al reported a case of a 27 year old female with 20 intra cranial meningiomas without evidence of neurofibromatosis. It is likely that all these cases belong together.

RECURRENCES IN MENINGIOMAS :

In this study, there were ten cases of recurrent meningiomas. Six of these cases were operated for the initial tumour at our Institute while four cases were referred from other hospitals. Three recurrent tumours occurred in the first two decades. One was a recurrent papillary meningioma in an

intraventricular location.

The recurrences among the adult patients were more in the men with five cases compared to two women. All the initial lesions were WHO grade I meningiomas while two lesions had progressed to become WHO grade II atypical meningiomas during recurrence. There was no site predilection among the recurrences.

Perry et al studied 44 cases of atypical and anaplastic meningiomas and concluded that sub total resections, male gender, and age less than 40 year are associated with a greater likelihood of recurrences[2]. They also noted a higher incidence of recurrent meningiomas being located in the convexities.

MORBID ANATOMY

Grossly most of the specimens of meningiomas received during the study were fragmented specimens measuring about 10 cc on an average. The largest specimen was an atypical meningioma that measured about 8cms in diameter. Some cases were received with the attached duramater and three cases with the adjacent involved bone. All the bony specimens showed thickening as evidence of hyperostosis.

The cut surface varied according to the histological type. Most cases were firm and lobulated, tan to brown – grey in colour. Microcystic meningiomas demonstrated tiny cysts even grossly, while the psammomatous meningiomas had a gritty quality while cutting. Metaplastic meningiomas with xanthomatous changes had a pale yellow hue on the cut surface. There was a single case of a cystic meningioma with a mural nodule that proved to be a meningothelial meningioma on histopathological examination.

Over 75% of the cases in this study had undergone a sub total resection particularly when the

site involved was difficult to access like the sphenoid wing or posterior cranial fossa.

HISTOPATHOLOGICAL VARIANTS OF MENINGIOMAS

The WHO 2000 classification of meningiomas has defined fifteen variants of which nine are classified as WHO grade I tumours. Three WHO grade II tumours and three WHO grade III tumours are listed[28].

Of the 342 meningiomas studied during a five year period all the variants of the new classification were seen with the exception of clear cell meningiomas. WHO grade I meningiomas formed the largest category with 320 cases. Among these, meningothelial meningiomas were the most common type accounting for 128 cases.

The frontal lobes were the site most frequently involved by them, with 21 cases, followed by the parietal lobe with 16 cases, and sphenoid wing with 12 cases.

Among the intraventricular meningiomas, meningothelial variant accounted for a majority of the cases.

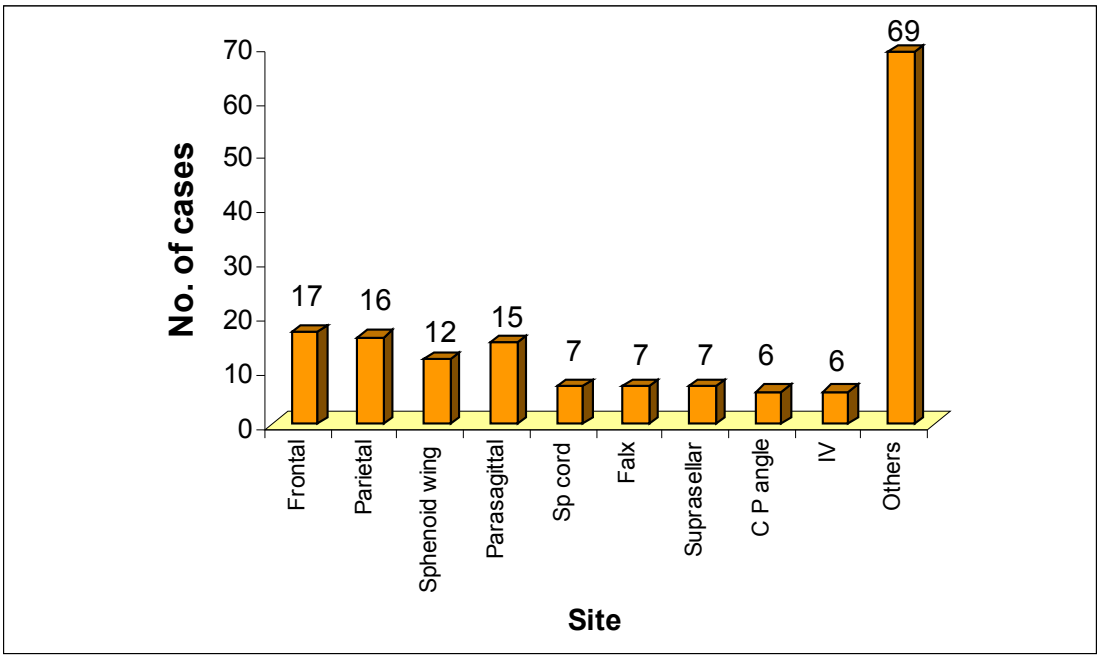
From the classic Russell and Rubinstein textbook, Pathology of tumours of the nervous system, meningothelial meningioma have been the most common category. A review of 936 patients by Jaackelainen et al showed 43% of the cases to be syncytial meningiomas.

Table 11

HISTOLOGICAL VARIANTS

Variants	No. of Cases	Percentage
Meningothelial	128	37.4%
Fibrous	77	22.51%
Transitional	47	13.74%
Psammomatous	33	9.64%
Angiomatous	23	6.72%
Lymphoplasmacytic	3	0.87%
Microcystic	4	1.16%
Secretory	1	0.29%
Metaplastic	5	1.46%
Atypical	10	2.92%
Chordoid	1	0.29%
Papillary	6	1.75%
Rhabdoid	3	0.87%
Anaplastic	1	0.29%

SITE WISE DISTRIBUTION OF MENINGOTHELIAL MENINGIOMAS



The histology of most of these cases were typical with whorls, sheets and nests of ill – refined meningothelial cells having abundant cytoplasm and vestibular nuclei with marginated chromatin.

Though a few cases exhibited pleomorphic nuclei, those without increased mitotic activity were still classified as meningothelial grade I meningiomas.

Fibrous meningiomas were the second most common subtype. The frequency of fibrous meningiomas were found to increase with age. There were 25 fibrous examples among the 104 meningiomas reported in the 5th decade, and 10 cases out of the reported 47 meningiomas in the 6th decade.

Shah et al[42] have noted a slight increase in the frequency of fibrous meningiomas with age.

Meningothelial and fibrous meningiomas were found in equal frequency in the cerebello – pontine angles.

Literature states that childhood meningiomas show a predominance of papillary, clear cell or

chordoid sub types[10].

Out of the 6 papillary meningiomas in our series three examples occurred in the first three decades. One case presented with a recurrence, while the other two were lost to follow up.

A single case of chordoid meningioma was reported in a 46 years old male in the regions of the clivus. The tumour had eroded the posterior clinodal process and a partial excision was performed. Through the patient had a microcytic anaemia be did not have hypergamma globulinemia or associated castheman's disease. The patient expired two days after surgery.

Kepes et al described the chordoid variant of meningioma for the first time, and also stated that administration of steroids resulted in complete resolution of symptoms[23]. Couce et al in a large series of 42 patient observed that none of their cases had systemic manifestations and that the average age of their patient was 47.4 years[4].

Recurrence rate is high among chordoid meningiomas which are classified as WHO grade II tumours. A possible explanation could be related to the mucoid quality of its stroma which mechanically facilitates the spread of the neoplastic cells. Recurrent tumours usually have an mucin rich chordoid pattern.

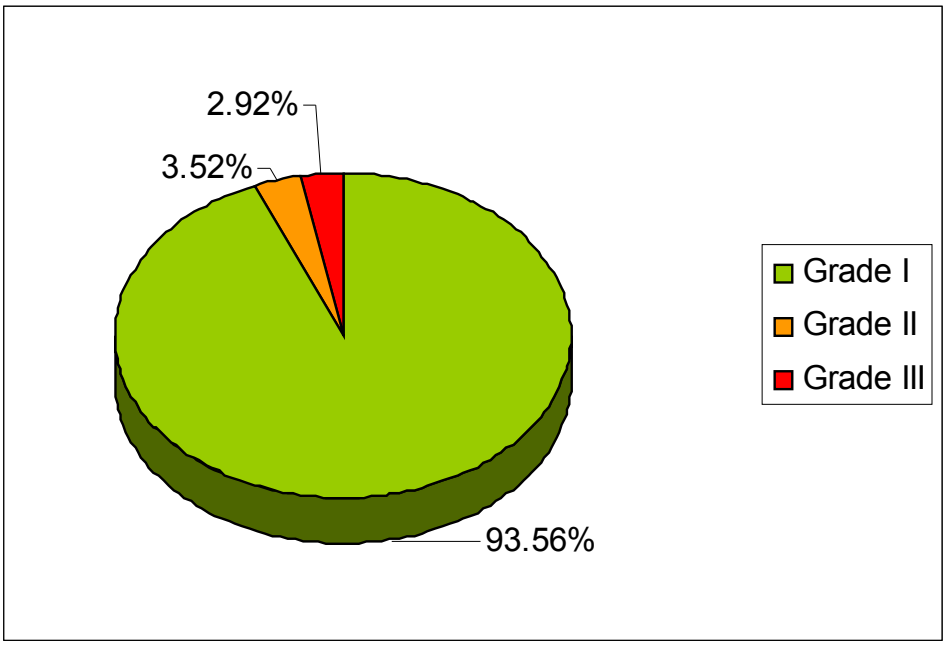
Three cases of rhabdoid meningiomas WHO grade III were reported. Two were in their sixties while the other was a young 17 years old female. The latter patient died a day after surgery following a massive epileptic attack and aspiration.

Rhabdoid meningiomas are WHO grade III and have an aggressive clinical course. Their histology is similar to rhabdoid tumours elsewhere in the body. There were large areas of necrosis, extreme cytological atypia and high mitotic counts.

Uncommonly, rhabdoid meningiomas without malignant features have been described, the

behaviour of these tumours being undetermined as yet.

MENINGIOMA - GRADES



Atypical and Anaplastic Meningioma

There were 10 atypical meningioma under WHO grade II meningiomas, forming 2.92% of the total. Two of these were recurrent lesions. They were seen with a slightly higher frequency among men. No specific site predilection was noted. The age groups commonly involved were the fifth and sixth decades.

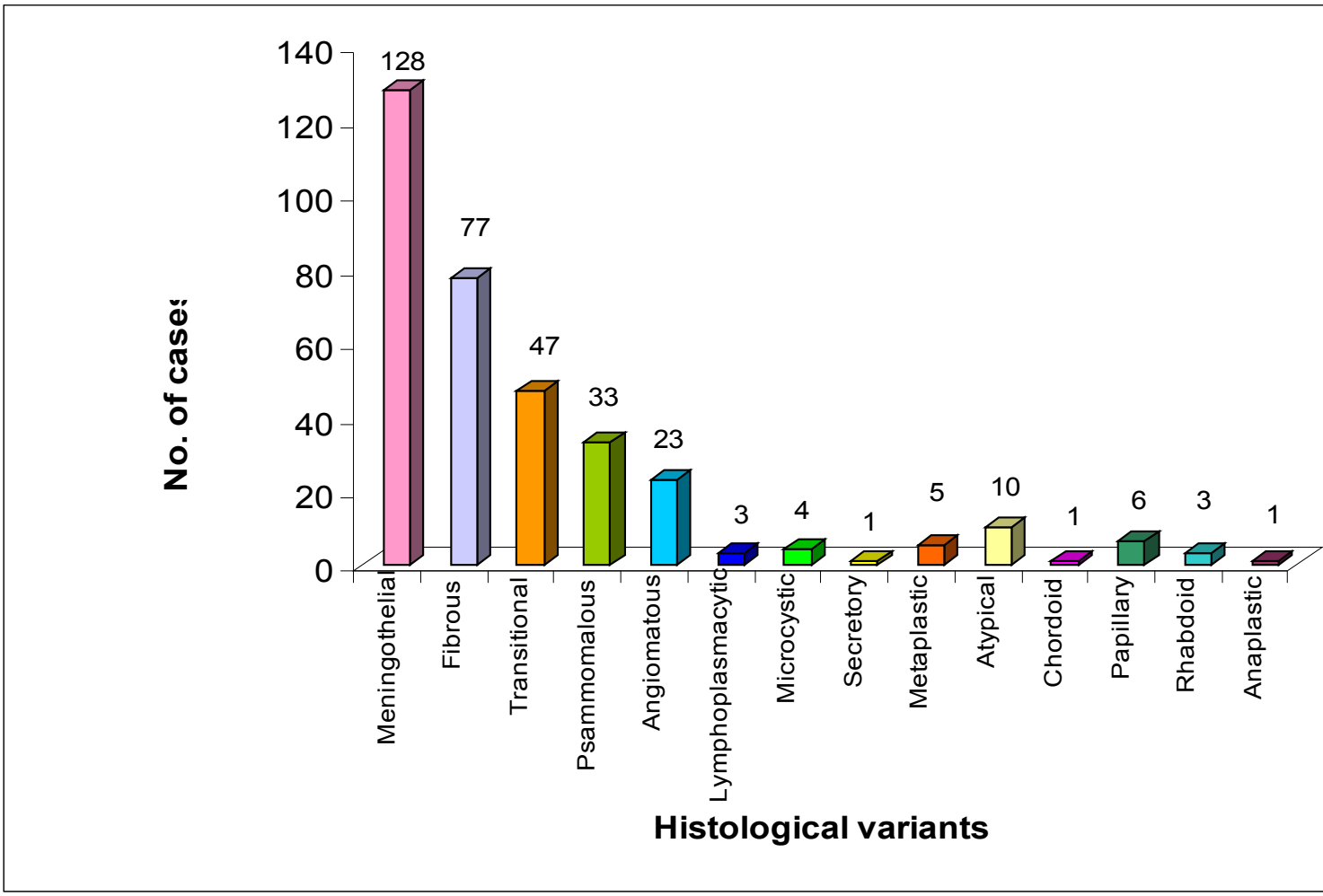
A single case of anaplastic meningioma was reported in a 16 year old girl in the posterior cranial fossa. The mitotic count was 30/hpf and the course was aggressive with the patient succumbing to the lesion 2 months after surgery.

Malhotra et al reported an incidence of 3.8% (5 out of 130) papillary meningiomas[42]. Atypical meningiomas accounted for 8.3% (32 out of 382) in a series reported by Joseph et al and only one malignant meningioma was encountered.

Perry et al stated the two most use ful criteria for predicting recurrences (Mayo Clinic Series) were brain invasion and presence of mitotic activity (at least 4/ hpf).

Mc Carthy et al in their large series of 9000 cases determined that important prognostic factors for benign tumours included age at diagnosis, tumor size, surgical treatment and radiation therapy.

DISTRIBUTION OF THE HISTOLOGICAL VARIANTS OF MENINGIOMAS



INFERENCE

Benign meningiomas of various histological types formed the vast majority of cases in this

study. Meningiomas were more common in women than men and show an increasing trend with age at least upto the fifth decade. Child hood meningiomas alone show a male preponderance. These tumours are located supratentorially in most cases.

Among the benign meningiomas meningothelial examples are the most common followed by fibrous, transitional and other variants. Histological grade, grade of surgical resection and adjuvant therapy influence survival in most cases.

Fig.1, Meningothelial meningioma – whorled cut surface

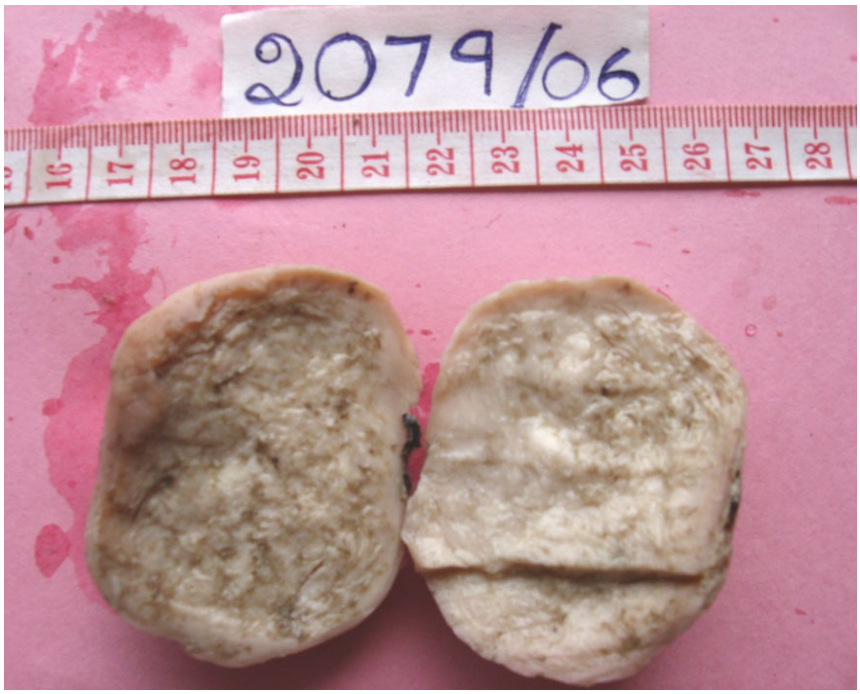


Fig.2, Fibrous Meningioma

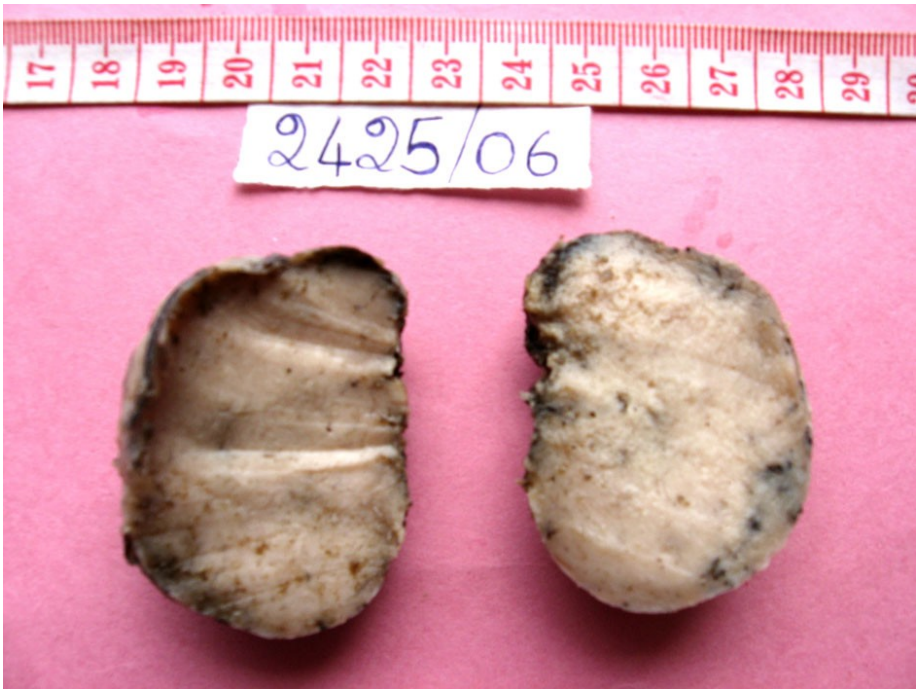


Fig.3, Transitional Meningioma

Fig.4, Lymphoplasmacytic Meningioma
Homogenous gray brown cut surface

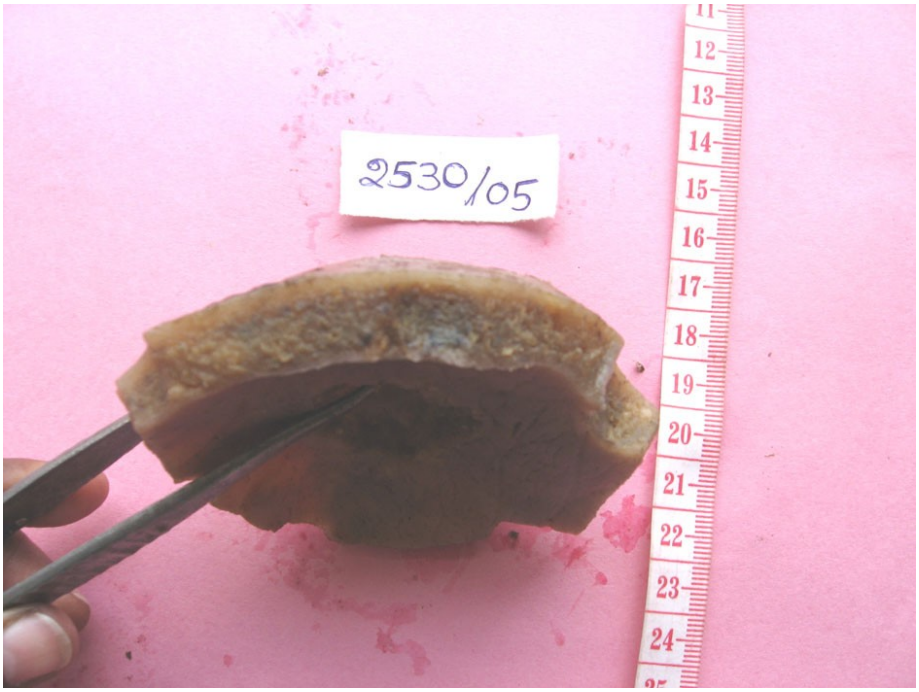


Fig.5, Hyperostotic Parietal Bone

Fig.6, Atypical Meningioma – Showing areas of necrosis

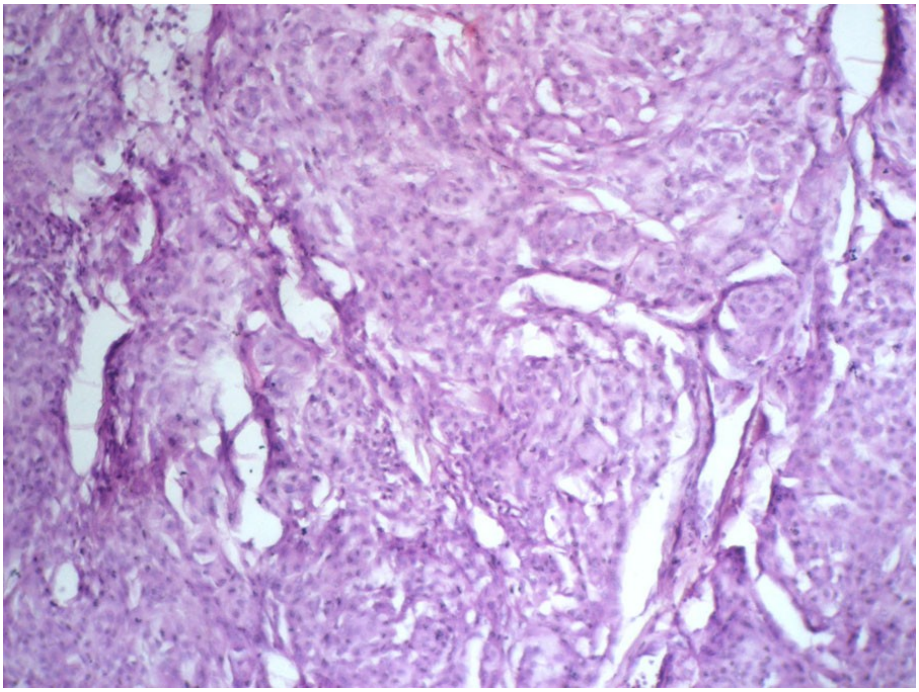


Fig.7, Meningothelial Meningioma – Syncytial Whorls of Meningothelial Cells H&E (x 100)

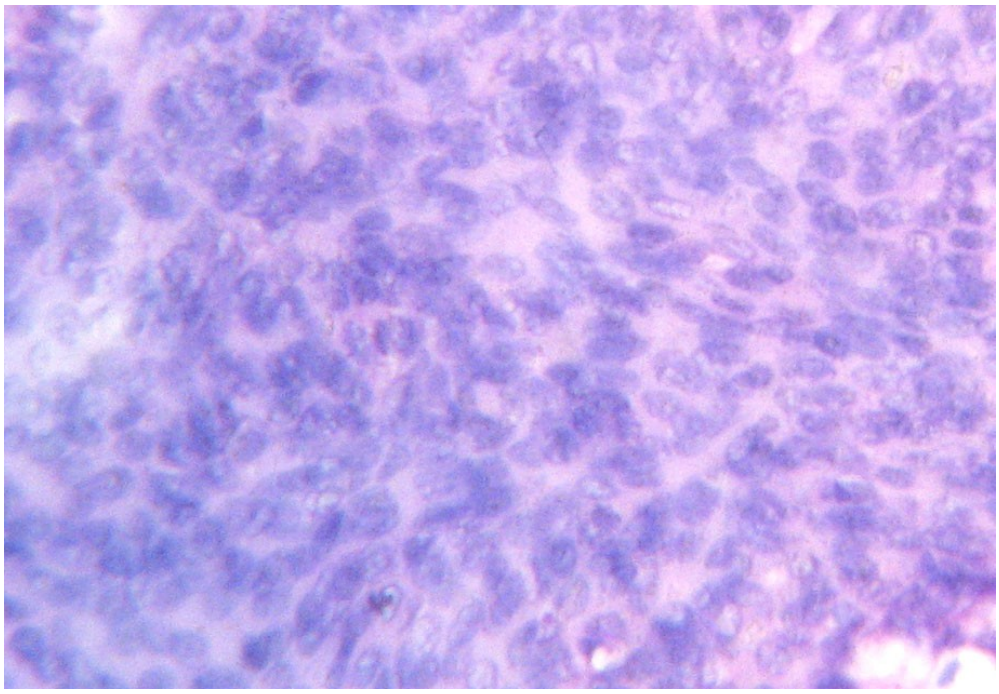


Fig.8, Meningothelial Meningioma
Vesicular nuclei with marginated chromatin. H&E (x 400)

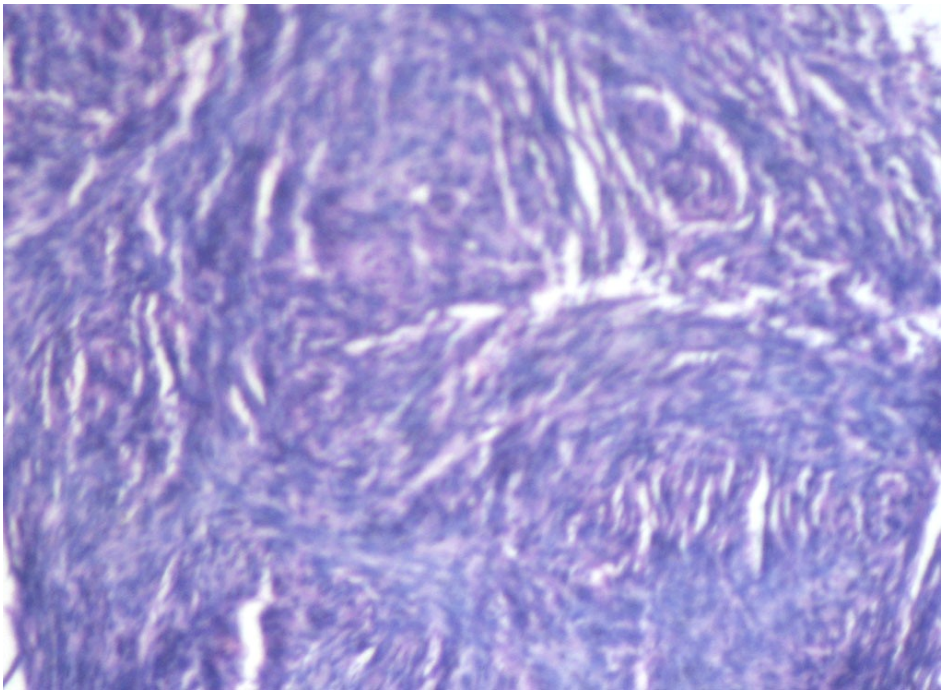


Fig.9, Fibrous Meningioma - Interlacing Fascicles of thin spindle shaped cells H & E (x 100)

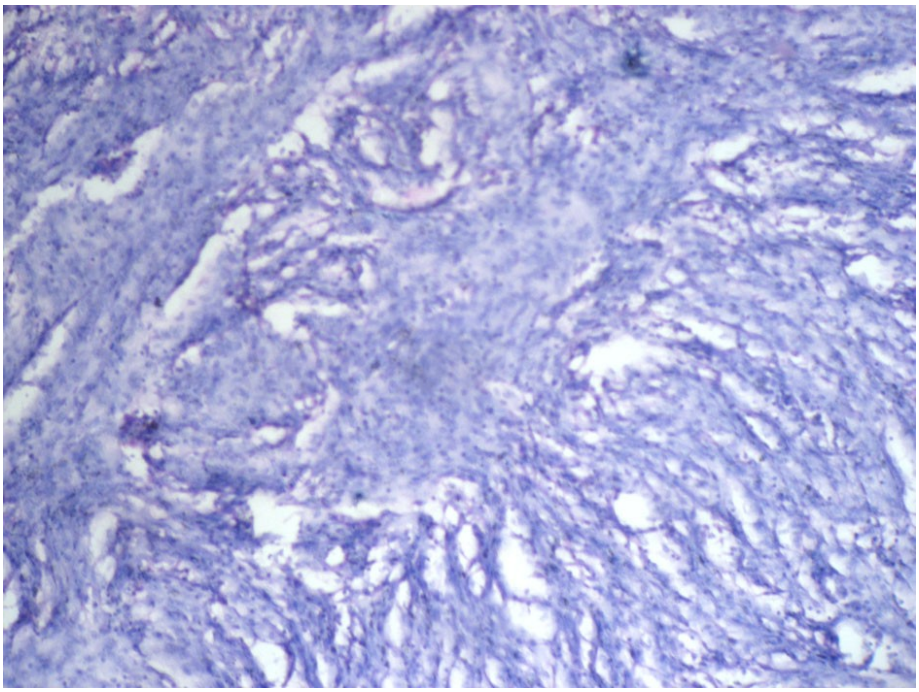


Fig.10, Transitional Meningioma – Both syncytial islands and fibrous areas H & E (x 100)

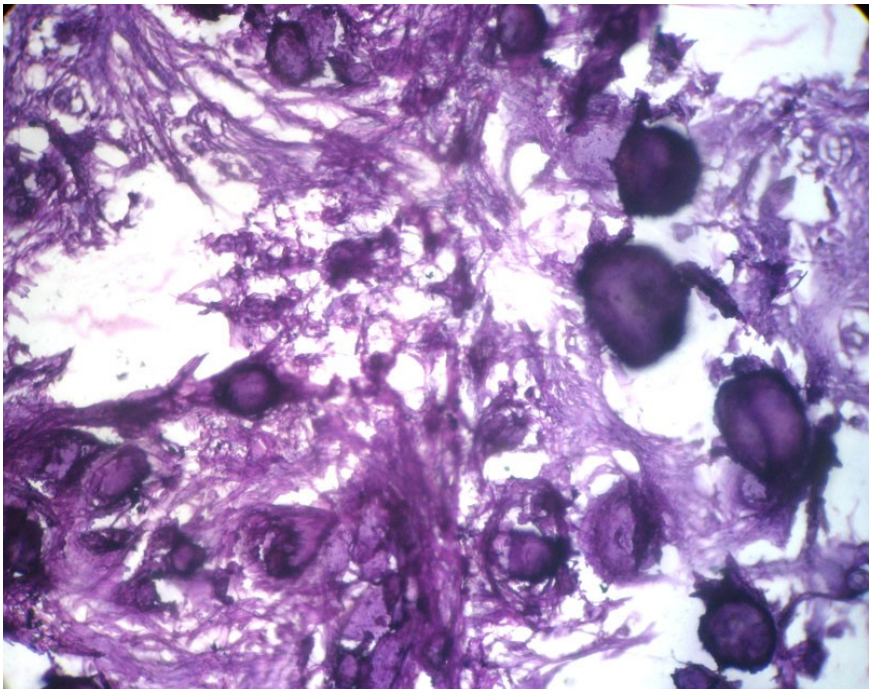


Fig.11, Psammomatous Meningioma – Numerous psammoma bodies H & E (x 100)

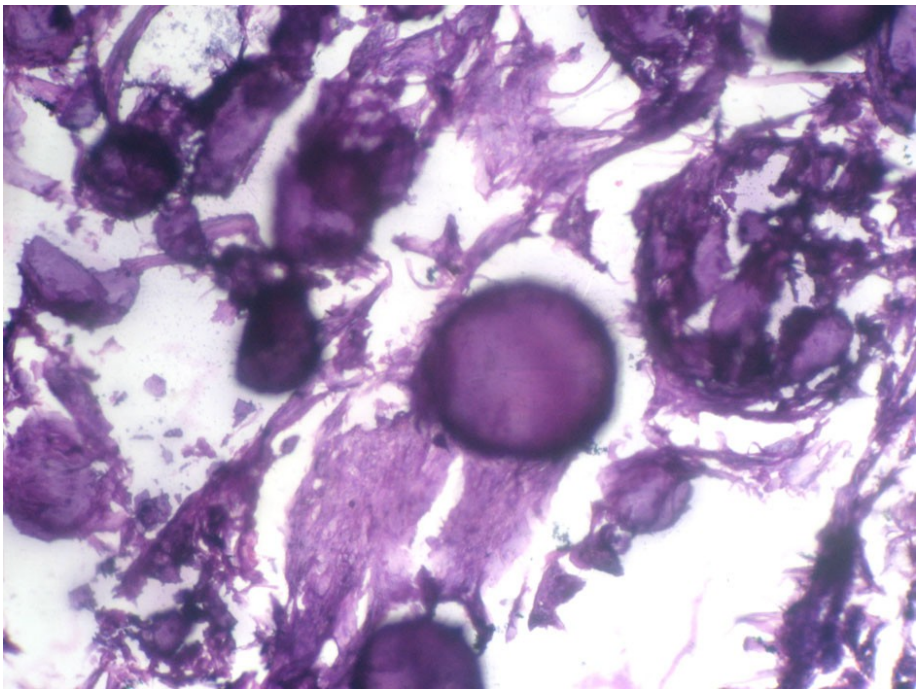


Fig.12, Psammoma bodies - H & E (x 400)

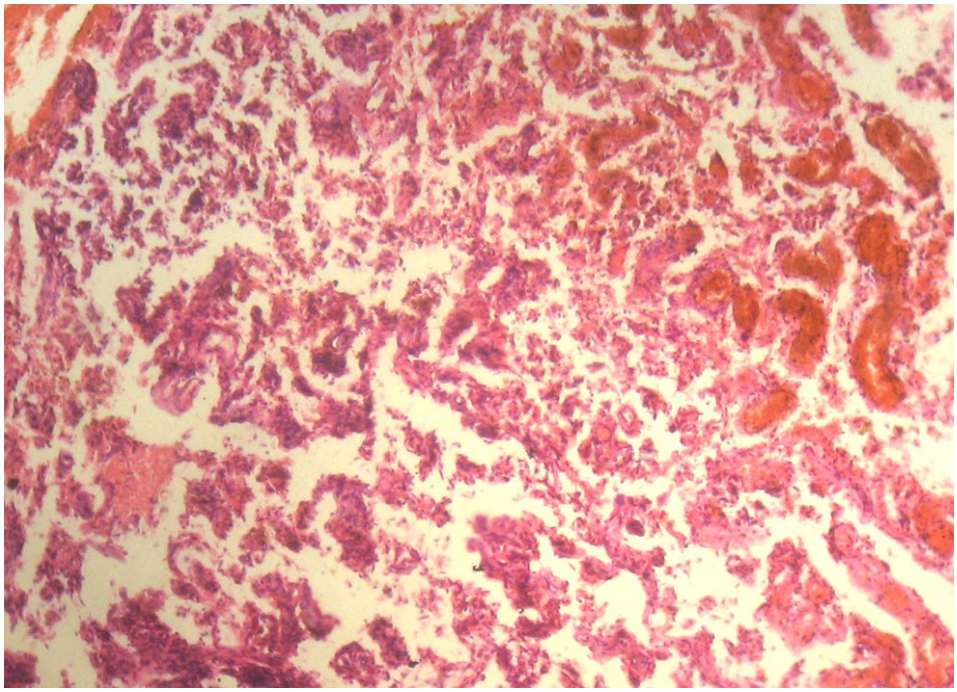


Fig.13, Angiomatous Meningioma – Numerous blood vessels interspersed among the meningothelial cells - H & E (x 100)

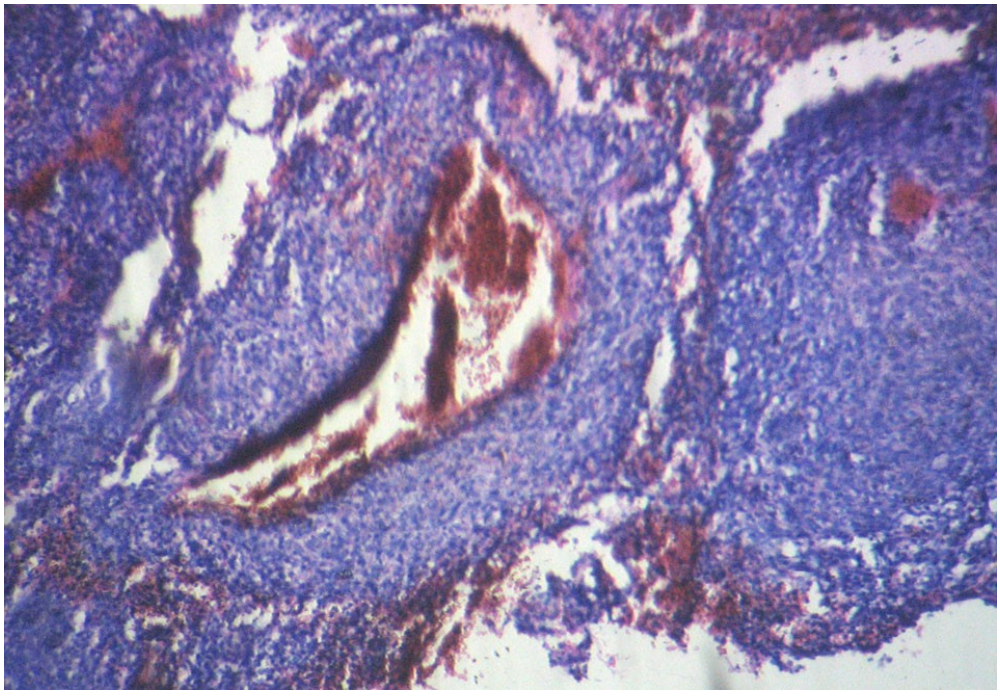


Fig.14, Angiomatous Meningioma – Large caliber vessels H & E (x 100)

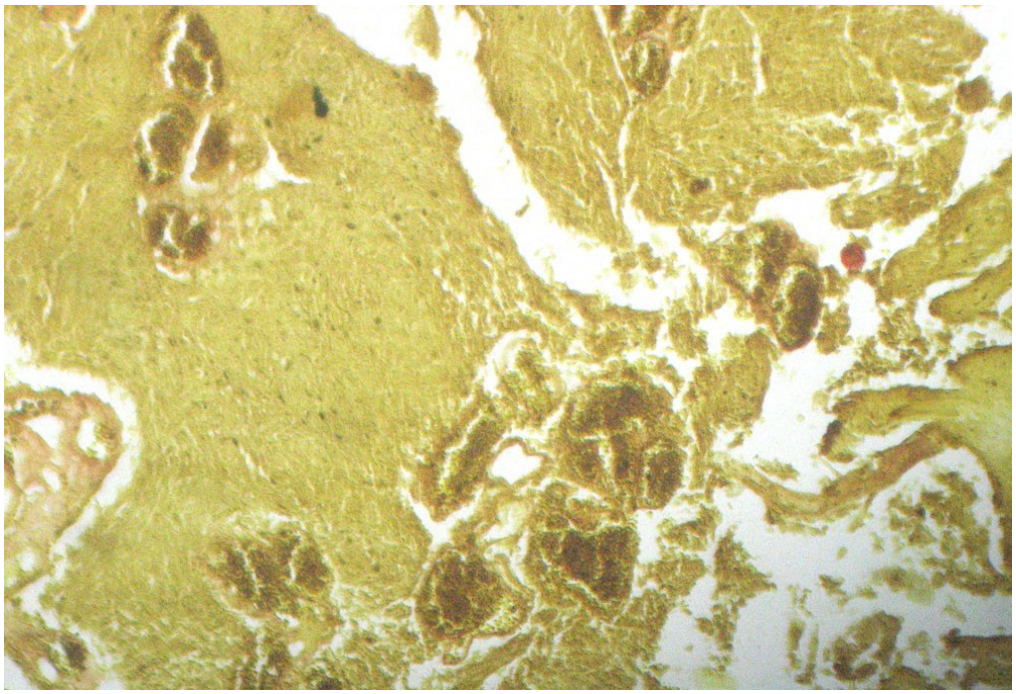


Fig.15, Angiomatous Meningioma – Tumour vasculature showing lack of elastic laminae. Verhoeff Van Gieson (x 100)

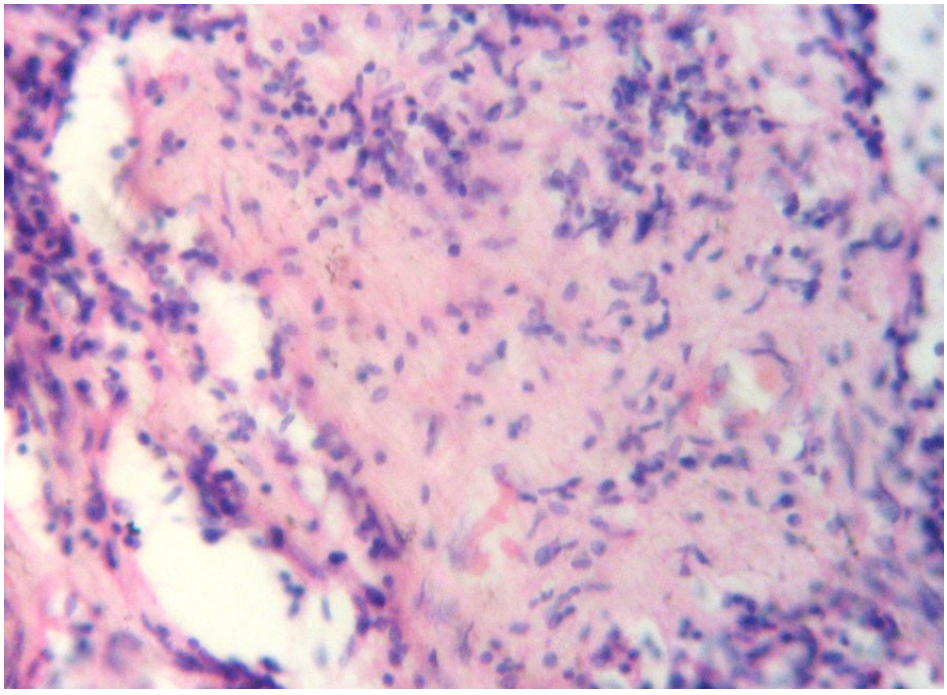


Fig.16, Lymphoplasmacytic meningioma
Showing dense lymphoplasmacytic infiltrate. H & E (x 400)

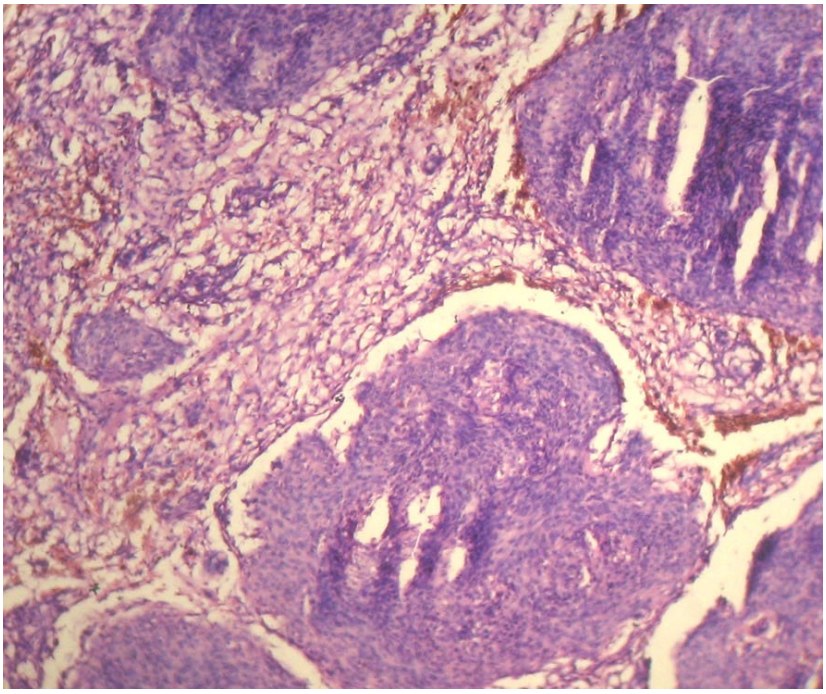


Fig.17, Metaplastic Meningioma – Islands of meningothelial cells separated by mature adipose tissue. H & E (x 100)

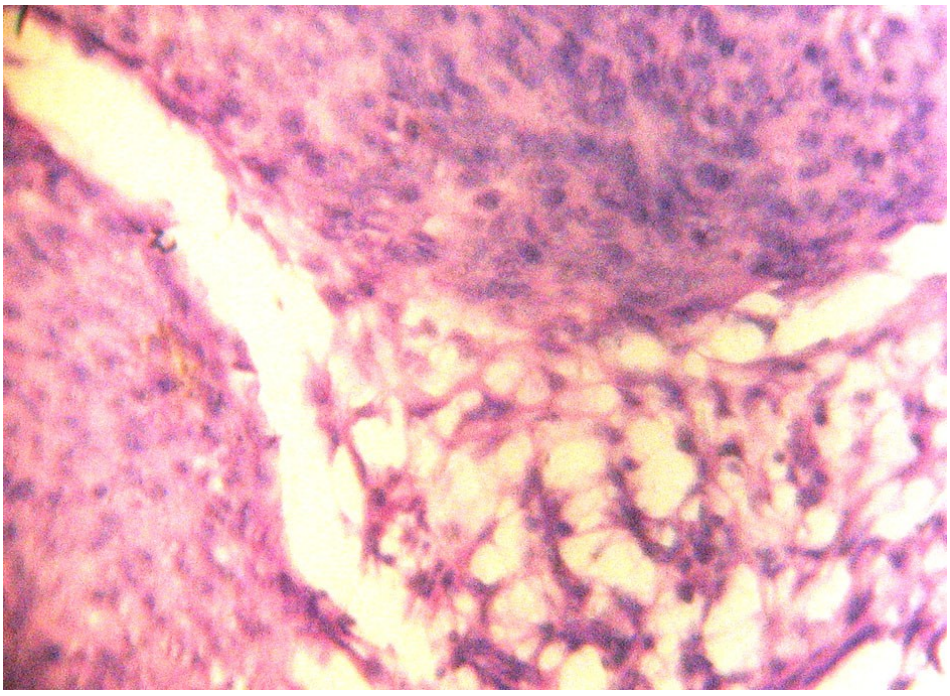


Fig.18, Metaplastic Meningioma – Lipomatous metaplasia. H & E (x 400)

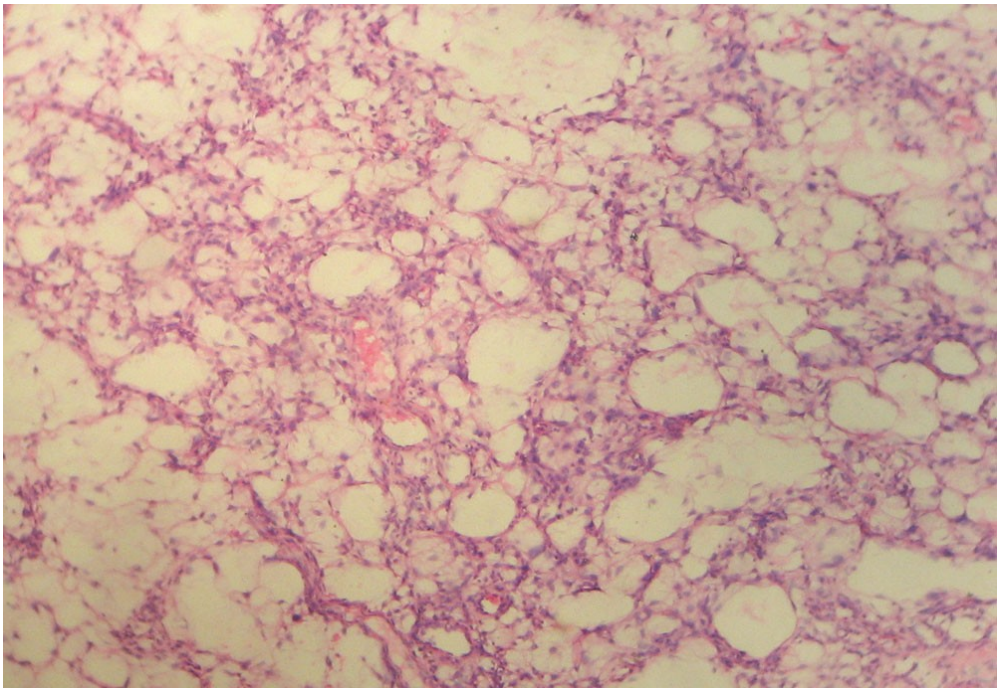


Fig.19, Microcystic Meningioma – Numerous cystic spaces of various sizes H & E (x 100)

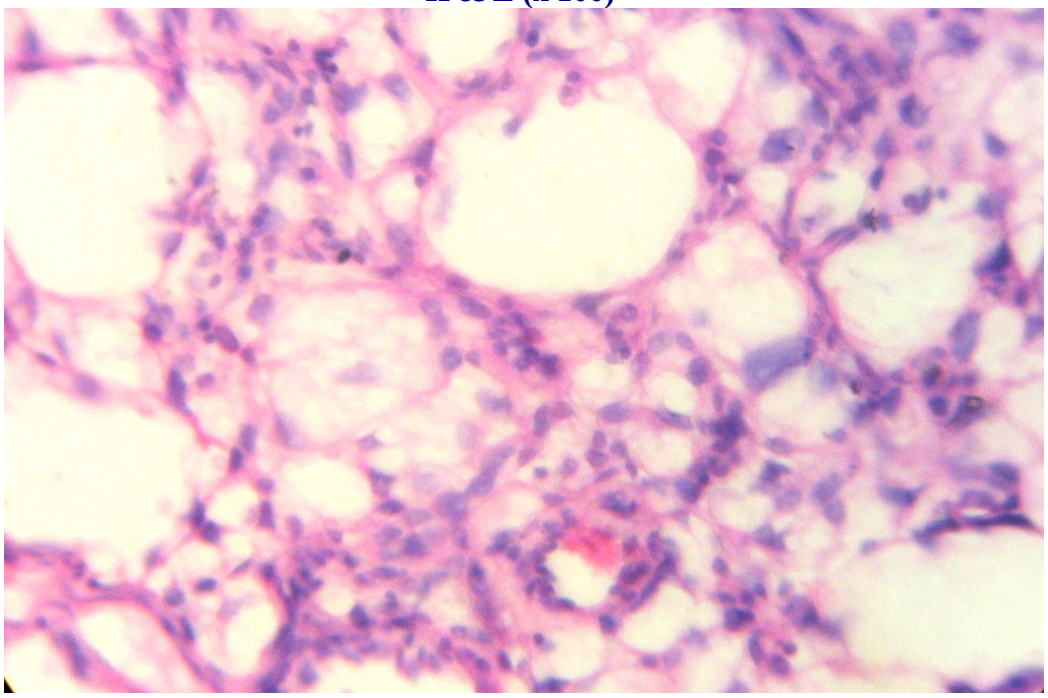


Fig.20, Microcystic Meningioma H & E (x 400)

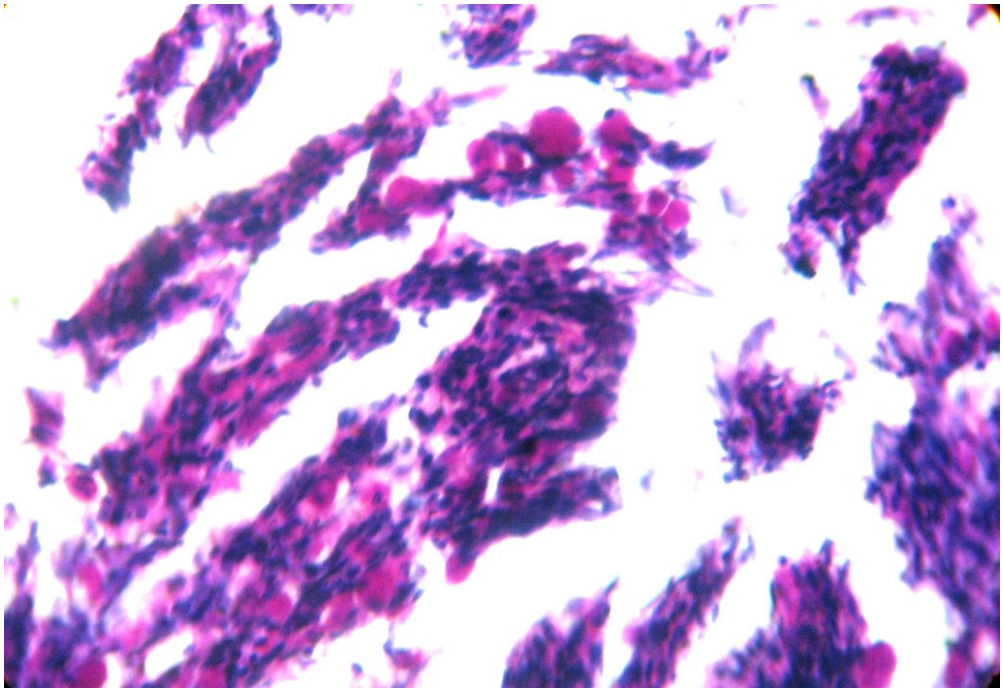


Fig.21, Secretory Meningioma – PAS positive globules among small meningothelial cells groups PAS (x 100)

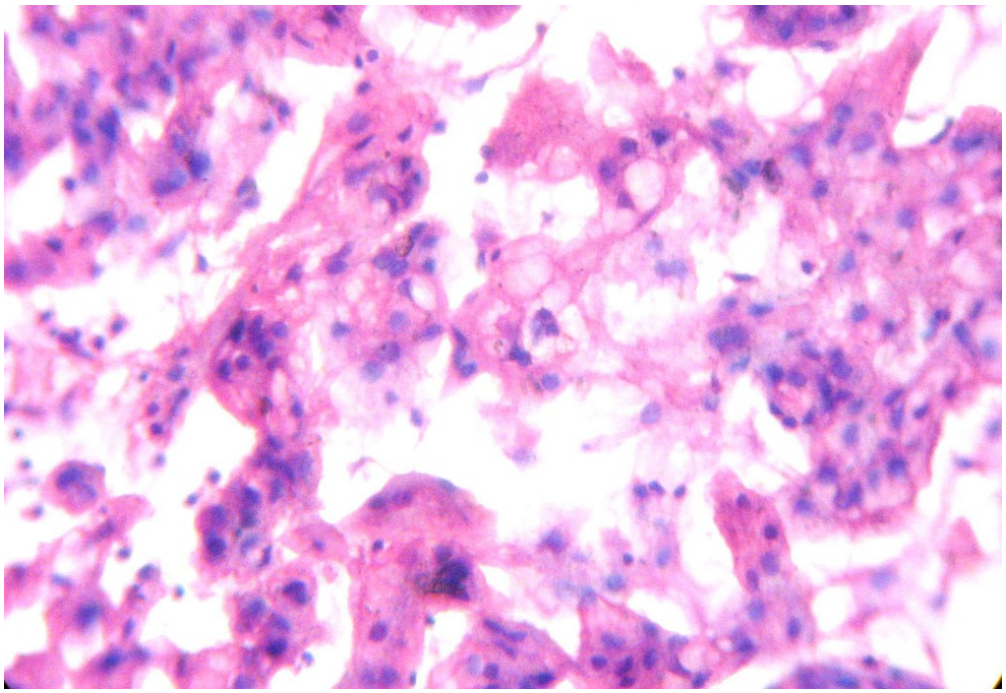


Fig.22, Chordoid Meningioma – Cords of cells with abundant cytoplasm H & E (x 100)

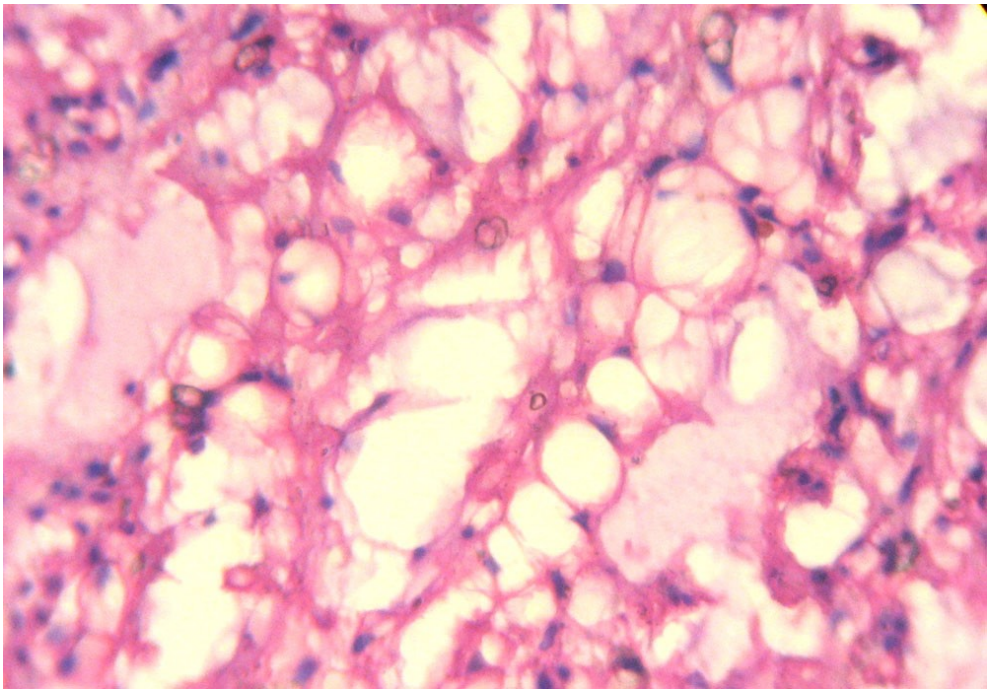


Fig.23, Chordoid Meningioma – Physaliferous cells with "bubbly" cytoplasm
H & E (x 400)

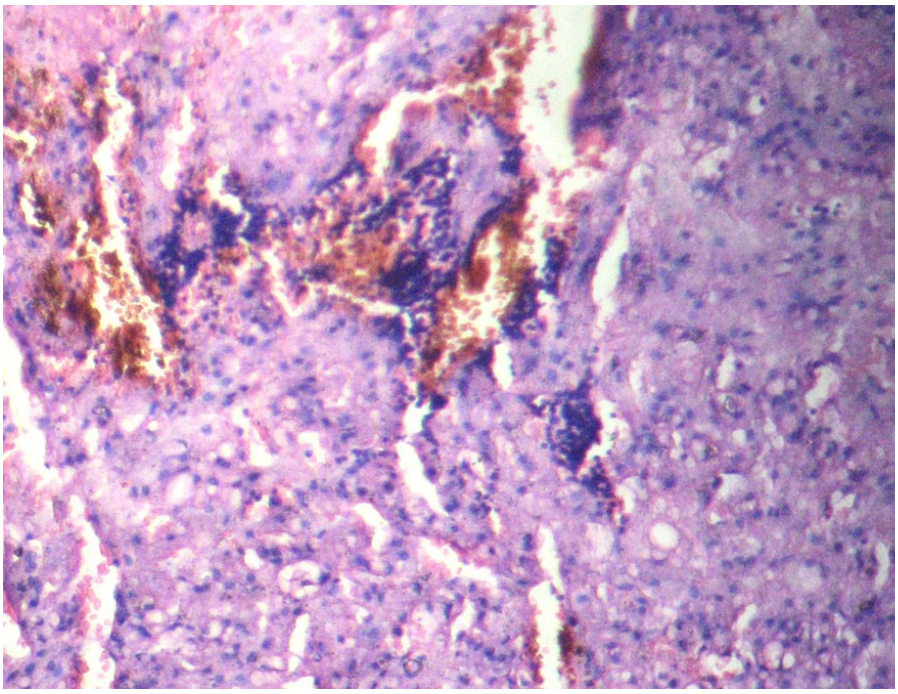


Fig.24, Chordoid Meningioma – Showing lymphoplasmacytic infiltration
H & E (x 100)

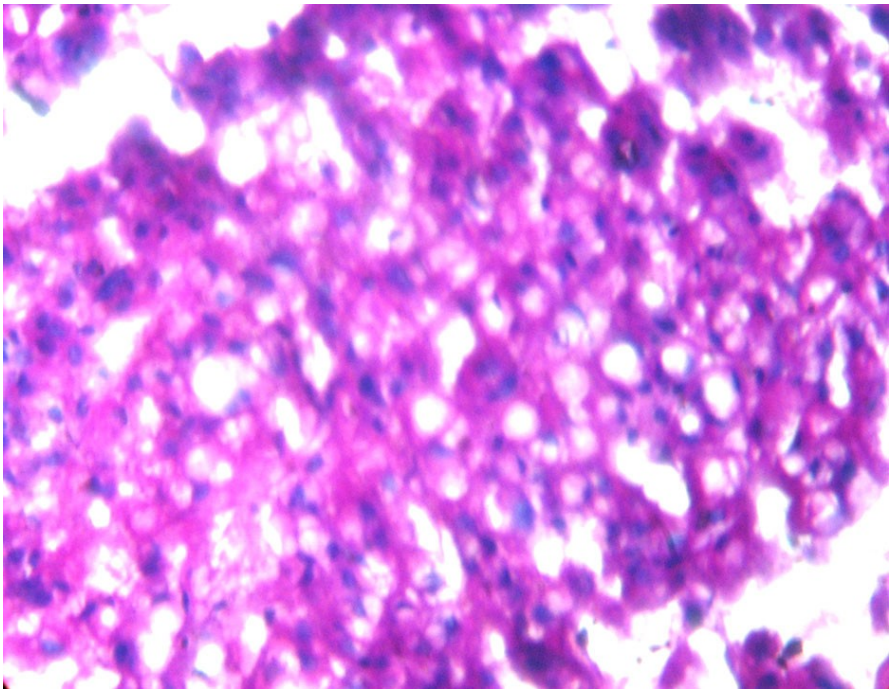


Fig.25, Chordoid Meningioma – Periodic Acid Schiff (x 100)

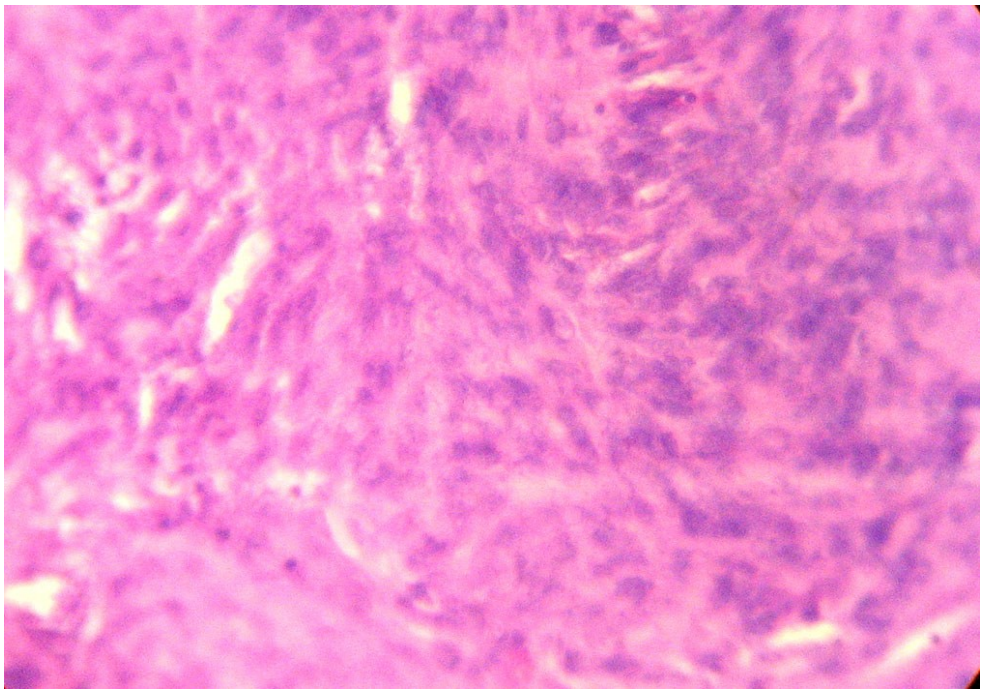


Fig.26, Atypical Meningioma – Sheet like arrangement of cells
H & E (x 100)

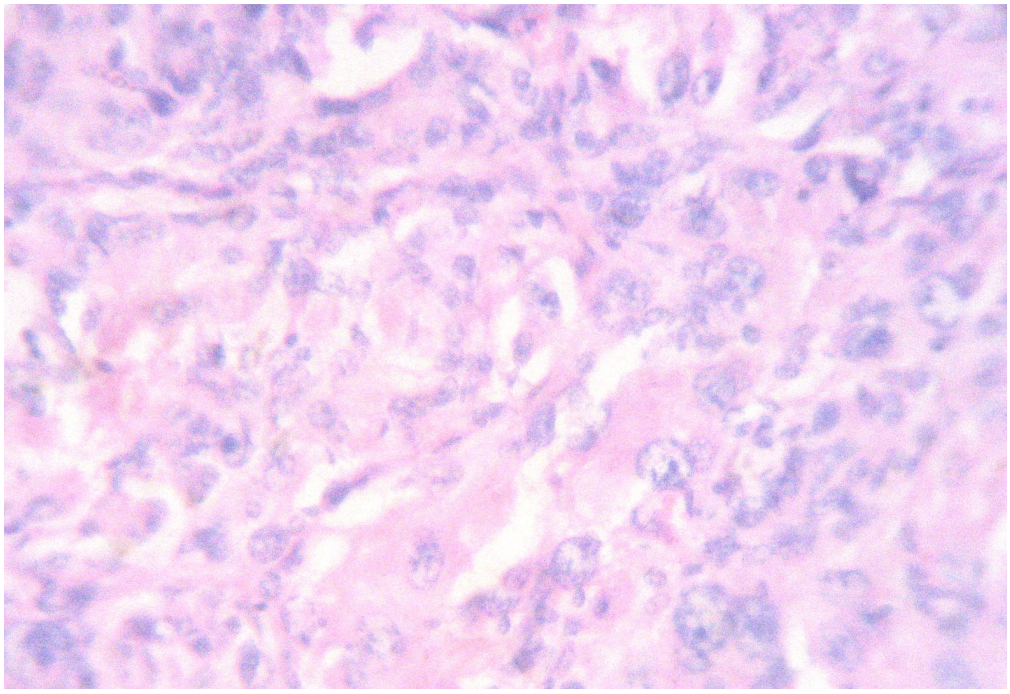


Fig.27, Rhabdoid Meningioma – Pleomorphic cells with abundant cytoplasm and multinucleation H & E (x 400)

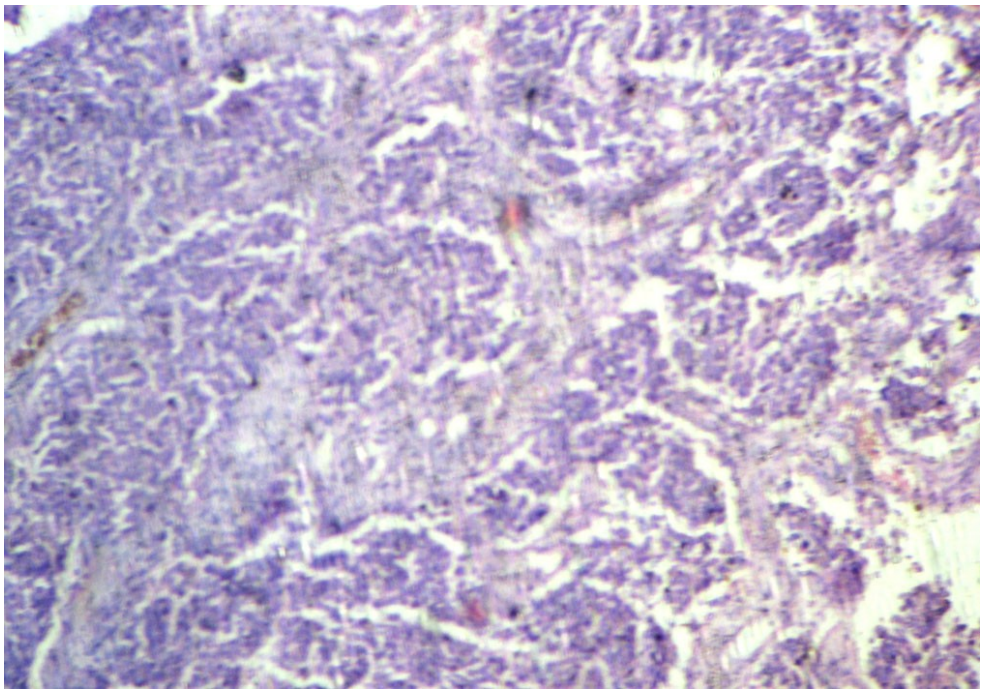


Fig.28, Papillary Meningioma – Cellular neoplasm showing small papillae

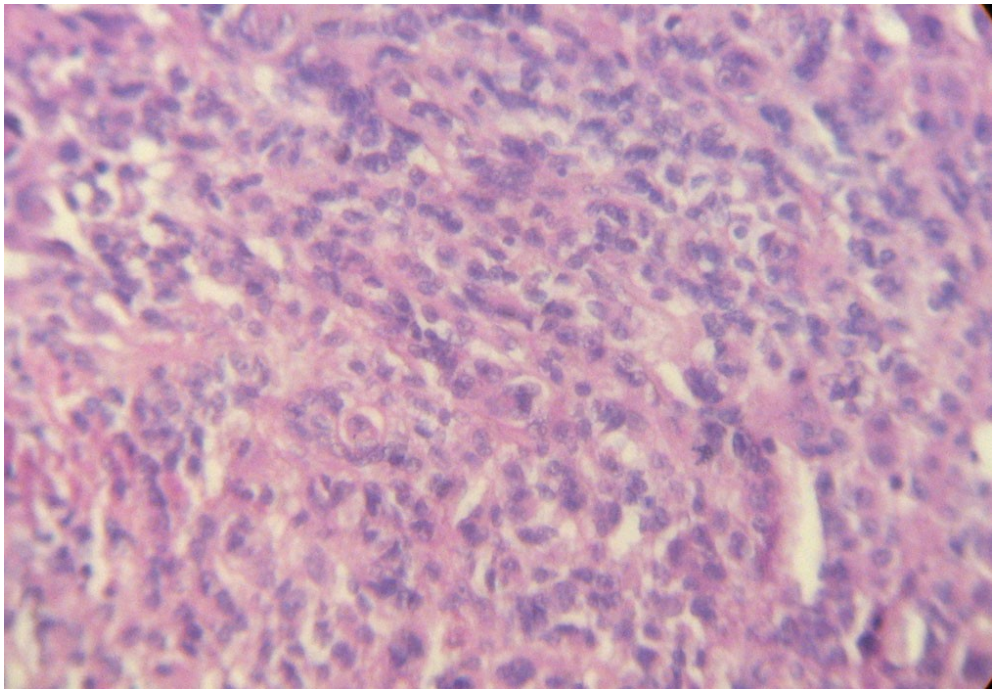


Fig.29, Anaplastic Meningioma – Sheet like arrangement of pleomorphic cells H & E (x 100)

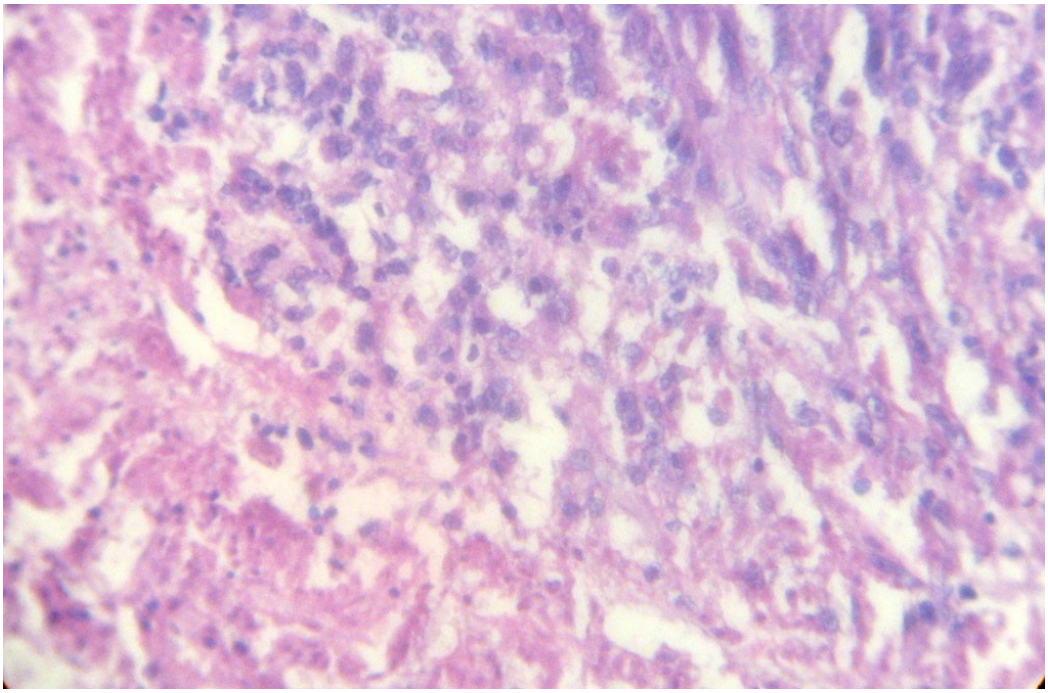


Fig.30, Anaplastic Meningioma – Showing areas of necrosis H & E (x 100)

SUMMARY AND CONCLUSION

The present study included 342 cases of meningiomas resected during the period from January 2001 to August 2006. The salient features observed in this study are :

1. Meningiomas constitute 17.57% of the total 1946 central nervous system tumours received during this period

2. They formed the second largest category after gliomas.

3. The youngest and oldest patients in the study were 5 years and 78 years respectively with a mean age of 46 years.

4. Of 342 cases in the study, females constituted 212 cases (61.98%) and males 130 cases (38.01%).

5. The peak incidence was in the fifth decade with 104 cases constituting 30.40% of the total number of cases.

6. There were 10 meningiomas in the first decade, with a male predominance 9 cases were males while there was only a single female patient.

7. The most common site in the present study were the frontal lobes with 63 cases, followed by the parietal lobes with 62 cases.

8. Intra ventricular meningiomas which are considered rare tumours numbered 10 accounting for 2.92% of the cases.

9. The most common presenting symptom was headache accompanied by nausea and vomiting. Recent onset seizures were the next most common symptom/

10. Irrespective of the histological type, the CT findings of meningiomas are usually hyperdense to isodense, enhancing well with contrast.

11. Benign WHO grade I meningiomas formed the overwhelming majority with 320 cases forming 93.56% of the total. Grade II meningiomas numbered 11, forming 3.52% while grade III meningiomas numbered 10 (2.92%).

12. Meningothelial meningiomas were 128 in number formed the most common histopathological type, (37.42%) followed by fibrous meningiomas which were 77 in number (22.51%).

13. Rare variants like secretory meningoma (1), chordoid meningioma (1), lymphoplasmacytic meningiomas (3), microcystic meningiomas (4) and rhabdoid meningiomas (3) were encountered during the study

The diagnosis of meningiomas has always depended upon conventional histomorphological study of H & E stained sections. Categorization into various histological types and grading as per the guidelines of the 2000 WHO classification also is done by conventional methods. The prognosis and response to treatment of the various types of meningiomas well with the present system of grading. Ancillary techniques like immunohistochemistry and electronmicroscopy are usually not required unless the tumor is very undifferentiated and anaplastic.

A lot of research is being done with reference to meningiomas and a whole host of new molecular targets like claudin, bcl – 2 etc have been discovered. Every new molecular target discovered prompts the search for an antibody that may help either in the diagnosis or function as a therapeutic agent.The future holds promise that meningiomas may be treated by non invasive methods like targeted molecular therapy.

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MASTER CHART

S. No.	HPE No.	Age	Sex	Site	Histological type	grade
1.	3104/ 01	46	M	Para sagittal	Meningothelial	I
2.	3108/ 01	42	F	Spinal cord	Psammomatous	I
3.	3114/ 01	50	M	Foramen magnum	Meningothelial	I
4.	3119/ 01	45	F	Orbit	Meningothelial	I
5.	3129/ 01	60	F	Sphenoid wing	Fibrous	I
6.	3140/ 01	40	F	Supra sellar	Papillary	III
7.	3141/ 01	45	F	Para sagittal	Fibrous	I
8.	3154/ 01	65	M	Para sagittal	Meningothelial	I
9.	3165/ 01	45	M	Sphenoid wing	Meningothelial	I
10.	3167/ 01	22	M	Intra ventricular	Meningothelial	I
11.	3168/ 01	25	M	Posterior fossa	Psammomatous	I
12.	3172/ 01	45	F	CP Angle	Meningothelial	I
13.	3178/ 01	35	F	Frontal	Meningothelial	I
14.	3180/ 01	24	F	Spinal cord	Psammomatous	I
15.	3182/ 01	40	F	Para sagittal	Fibrous	I
16.	3192/ 01	50	M	Frontal	Meningothelial	I
17.	3199/ 01	50	M	Frontal	Fibrous	I
18.	3206/ 01	54	F	Frontal	Meningothelial	I
19.	3210/ 01	38	F	Para sagittal	Fibrous	I
20.	3212/ 01	50	M	Para sagittal	Fibrous	I
21.	3218/ 01	45	F	Orbit	Meningothelial	I
22.	3223/ 01	50	F	Parietal	Atypical	II
23.	3225/ 01	65	F	Para sagittal	Fibrous	I
24.	3231/ 01	06	M	Post Fossa	Angiomatous	I
25.	3234/ 01	20	F	Intraventricular	Meningothelial	I
26.	3237/ 01	28	F	Para sagittal	Meningothelial	I
27.	3240/ 01	45	M	Parietal	Meningothelial	I
28.	3243/ 01	35	F	Para sagittal	Meningothelial	I
29.	3247/ 01	60	M	Frontal	Fibrous	I
30.	3250/ 01	60	F	Orbit	Papillary	III
31.	3253/ 01	45	F	Parietal	Meningothelial	I
32.	3278/ 01	45	F	Parietal	Meningothelial	I
33.	3294/ 01	52	M	Parietal	Meningothelial	I
34.	3298/ 01	32	F	Spinal cord	Meningothelial	I
35.	3304/ 01	45	F	Spinal cord	Psammomatous	I
36.	3306/ 01	25	F	Temporal	Fibrous	I
37.	3307/ 01	35	F	Parietal	Meningothelial	I
38.	3320/ 01	40	M	Frontal	Meningothelial	I
39.	3338/ 01	46	F	Frontal	Meningothelial	I
40.	3342/ 01	42	M	Fontal	Meningothelial	I

S.	HPE No.	Age	Sex	Site	Histological type	grade

No.						
41.	3349/ 01	39	M	Parietal	Meningothelial	I
42.	3355/ 01	34	F	Frontal	Meningothelial	I
43.	3363/ 01	40	M	Parietal	Meningothelial	I
44.	3373/ 01	33	F	Spinal cord	Meningothelial	I
45.	3379/ 01	54	F	Parietal	Meningothelial	I
46.	3383/ 01	54	M	Para sagittal	Meningothelial	I
47.	3399/ 01	35	F	Temporal	Fibrous	I
48.	3402/ 01	65	F	Para sagittal	Fibrous	I
49.	3417/ 01	32	F	Spinal cord	Psammomatous	I
50.	3428/ 01	43	M	Parietal	Meningothelial	I
51.	3431/ 01	35	M	Falx	Meningothelial	I
52.	3436/ 01	42	F	CP Angle	Fibrous	I
53.	3444/ 01	30	F	Parietal	Meningothelial	I
54.	3467/ 01	22	F	CP angle	Meningothelial	I
55.	3474/ 01	50	F	Parietal	Meningothelial	I
56.	3484/ 01	17	F	Intraventricular	Papillary	III
57.	3501/ 01	35	F	Spinal cord	Meningothelial	I
58.	3503/ 01	40	F	Parietal	Meningothelial	I
59.	3532/02	40	M	Sphenoid wing	Meningothelial	I
60.	3548/02	25	F	CP angle	Psammomatous	I
61.	3568/02	30	F	Parietal	Meningothelial	I
62.	3577/02	35	F	Frontal	Meningothelial	I
63.	3579/02	72	M	Frontal	Meningothelial	I
64.	3582/02	38	F	Frontal	Angiomatous	I
65.	3585/02	18	F	Frontal	Angiomatous	I
66.	3587/02	55	M	Frontal	Meningothelial	I
67.	3599/02	34	F	Spinal cord	Psammomatous	I
68.	3604/02	65	F	Spinal cord	Transitional	I
69.	3625/02	30	F	Olfactory groove	Meningothelial	I
70.	3637/02	06	M	Spinal cord	Psammomatous	I
71.	3659/02	26	F	Spinal cord	Fibrous	I
72.	3669/02	38	M	Frontal	Meningothelial	I
73.	3666/02	53	M	Temporal	Angiomatous	I
74.	3667/02	50	F	CP angle	Meningothelial	I
75.	3672/02	41	M	Parietal	Meningothelial	I
76.	3681/02	49	M	Frontal	Meningothelial	I
77.	3683/02	50	M	Falx	Meningothelial	I
78.	3690/02	60	F	Spinal cord	Psammomatous	I
79.	3710/02	55	F	Parietal	Angiomatous	I
80.	3718/02	45	M	Sphenoid wing	Meningothelial	I

S. No.	HPE No.	Age	Sex	Site	Histological type	grade

81.	3744/ 02	35	F	Sphenoid wing	Transitional	I
82.	3750/ 02	55	F	Sphenoid wing	Meningothelial	I
83.	3754/ 02	20	F	Spinal cord	Psammomatous	I
84.	3765/ 02	30	F	Parietal	Meningothelial	I
85.	3781/ 02	43	F	Falx	Meningothelial	I
86.	3788/ 02	06	M	Parietal	Meningothelial	I
87.	3791/ 02	60	F	Frontal	Meningothelial	I
88.	3799/ 02	63	M	Parietal	Fibrous	I
89.	3803/ 02	24	F	Frontal	Fibrous	I
90.	3805/ 02	53	M	Temporal	Meningothelial	I
91.	3809/ 02	24	M	Intraventricular	Meningothelial	II
92.	3820/ 02	40	F	Pineal Region	Meningothelial	I
93.	3825/ 02	16	F	Post Fossa	Anaplastic	III
94.	3839/ 02	45	F	Falx	Meningothelial	I
95.	3841/ 02	70	F	Temporal	Meningothelial	I
96.	3865/ 02	45	F	Spinal cord	Meningothelial	I
97.	3868/ 02	54	M	Para sagittal	Fibrous	I
98.	3869/ 02	65	F	Spinal cord	Psammomatous	I
99.	3871/ 02	15	M	Sphenoid wing	Anaplastic	I
100.	3875/ 02	63	F	Sphenoid wing	Meningothelial	I
101.	3881/ 02	18	M	Sphenoid wing	Meningothelial	I
102.	3885/ 02	27	M	Sphenoid wing	Meningothelial	I
103.	3940/ 03	70	M	Frontal	Meningothelial	I
104.	3945/ 03	50	F	CP angle	Fibrous	I
105.	3954/ 03	10	M	Para sagittal	Fibrous	I
106.	3984/ 03	30	F	Posterior Fossa	Psammomatous	I
107.	3986/ 03	45	F	Frontal	Fibrous	I
108.	3988/ 03	35	F	Temporal	Meningothelial	I
109.	3992/ 03	54	M	Parietal	Transitional	I
110.	4003/ 03	42	F	Posterior Fossa	Meningothelial	I
111.	4013/ 03	45	M	Sphenoid wing	Meningothelial	I
112.	4022/ 03	40	F	Frontal	Meningothelial	I
113.	4028/ 03	05	F	Temporal	Papillary	III

114.	4035/ 03	30	F	Temporal	Psammomatous	I
115.	4045/ 03	29	M	Sphenoid wing	Meningothelial	I
116.	4050/ 03	45	M	Temporal	Meningothelial	I
117.	4058/ 03	45	F	CP angle	Meningothelial	I
118.	4059/ 03	52	F	Frontal	Meningothelial	I
119.	4069/ 03	39	M	Frontal	Meningothelial	I
120.	4070/ 03	33	M	Frontal	Meningothelial	I
121.	4073/ 03	40	M	Parietal	Meningothelial	I
122.	4094/ 03	50	F	Occipital	Atypical	II

S. No.	HPE No.	Age	Sex	Site	Histological type	grade
123.	4096/ 03	50	F	Posterior Fossa	Transitional	I
124.	4099/ 03	50	F	Frontal	Transitional	I
125.	4100/ 03	35	M	Frontal	Psammomatous	I
126.	4101/ 03	45	F	Suprasellar	Meningothelial	I
127.	4108/ 03	45	F	Parietal	Transitional	I
128.	4110/ 03	35	F	Suprasellar	Meningothelial	I
129.	4113/ 03	37	M	Trigonal	Transitional	I
130.	4123/ 03	45	F	Parietal	Meningothelial	I
131.	4128/ 03	63	M	Suprasellar	Meningothelial	I
132.	4132/ 03	10	M	Frontal	Metaplastic	I
133.	4133/ 03	62	M	Frontal	Microcystic	I
134.	4144/ 03	46	M	Parietal	Meningothelial	I
135.	4148/ 03	50	M	Spinal cord	Angiomatous	I

136.	4162/ 03	42	M	Sphenoid wing	Meningothelial	I
137.	4175/ 03	35	F	Olfactory groove	Fibrous	I
138.	4177/ 03	55	F	Frontal	Angiomatous	I
139.	4197/ 03	45	F	Falx	Fibrous	I
140.	4213/ 03	40	F	Posterior Fossa	Transitional	I
141.	4217/ 03	32	F	Parietal	Transitional	I
142.	4219/ 03	35	F	Frontal	Fibrous	I
143.	4236/ 03	26	F	Frontal	Meningothelial	I
144.	4239/ 03	30	M	Falx	Meningothelial	I
145.	4242/ 03	35	M	Cerebellar	Fibrous	I
146.	4248/ 03	50	F	Falx	Meningothelial	I
147.	4259/ 03	65	F	Parietal	Fibrous	I
148.	4265/ 03	22	F	Frontal	Meningothelial	I
149.	4268/ 03	40	M	Parietal	Transitional	I
150.	4279/03	35	F	Temporal	Fibrous	I
151.	4290/ 03	37	M	Sphenoid wing	Angiomatous	I
152.	4303/ 03	45	F	Intraventricular	Meningothelial	I
153.	4308/ 03	42	M	Sphenoid wing	Transitional	I
154.	4331/ 03	27	M	Posterior Fossa	Fibrous	I
155.	4353/ 03	40	F	Falx	Meningothelial	I
156.	4368/ 03	28	F	Intraventricular	Transitional	I
157.	4374/ 03	36	M	Suprasellar	Transitional	I
158.	4399/ 04	39	F	Spinal cord	Fibrous	I
159.	1324/ 04	60	F	Temporal	Rhabdoid	III

160.	1329/ 04	33	F	Frontal	Fibrous	I
161.	1336/ 04	65	F	Intraventricular	Transitional	I
162.	1338/ 04	30	F	Spinal cord	Fibrous	I
163.	1372/ 04	43	F	Spinal cord	Psammomatous	I
164.	1376/ 04	45	F	Posterior Fossa	Fibrous	I

S. No.	HPE No.	Age	Sex	Site	Histological type	grade
165.	1385/ 04	28	F	Parietal	Angiomatous	I
166.	1388/ 04	40	F	Parietal	Atypical	II
167.	1405/ 04	32	M	Frontal	Transitional	I
168.	1406/ 04	49	M	Occipital	Fibrous	I
169.	1418/ 04	60	M	Spinal cord	Psammomatous	I
170.	1433/ 04	21	F	Cerebellar	Meningothelial	I
171.	1436/ 04	40	F	Frontal	Fibrous	I
172.	1441/ 04	24	F	Parietal	Transitional	I
173.	1465/ 04	42	F	Parietal	Fibrous	I
174.	1473/ 04	64	M	Posterior Fossa	Angiomatous	I
175.	1477/ 04	35	F	Spinal cord	Psammomatous	II
176.	1485/ 04	43	M	Frontal	Atypical	I
177.	1486/ 04	27	M	Posterior Fossa	Meningothelial	I
178.	1487/ 04	45	M	Corpus Callosum	Meningothelial	I
179.	1498/ 04	44	M	Sphenoid wing	Atypical	I
180.	1501/ 04	41	M	Intraventricular	Angiomatous	II
181.	1509/ 04	22	F	Frontal	Atypical	I

182.	1519/ 04	45	M	Parietal	Meningothelial	I
183.	1522/ 04	45	F	Spinal cord	Meningothelial	I
184.	1530/ 04	28	F	Parietal	Meningothelial	I
185.	1544/ 04	35	M	Spinal cord	Fibrous	I
186.	1545/ 04	32	F	Suprasellar	Meningothelial	I
187.	1547/ 04	44	M	Parietal	Transitional	I
188.	1553/ 04	10	M	Frontal	Metaplastic	I
189.	1557/ 04	65	M	CP Angle	Angiomatous	I
190.	1561/ 04	45	M	Frontal	Transitional	I
191.	1567/ 04	16	M	Sphenoid wing	Transitional	I
192.	1568/ 04	19	M	Parietal	Angiomatous	I
193.	1587/ 04	42	F	Parietal	Fibrous	I
194.	1592/ 04	36	M	Frontal	Meningothelial	I
195.	1601/ 04	54	M	Temporal	Microcystic	I
196.	1608/ 04	22	F	Suprasellar	Meningothelial	I
197.	1611/ 04	45	M	Falx	Fibrous	I
198.	1622/ 04	49	M	Parietal	Meningothelial	I
199.	1628/ 04	48	F	Spinal cord	Meningothelial	I
200.	1629/ 04	48	F	Frontal	Transitional	I
201.	1649/ 04	43	M	Parietal	Microcystic	I
202.	1654/ 04	55	F	CP angle	Meningothelial	I
203.	1656/ 04	45	F	Pineal Sol	Fibrous	I
204.	1658/ 04	38	M	Parietal	Transitional	I
205.	1626/ 04	48	M	Frontal	Angiomatous	I

206.	1680/ 04	28	F	Parietal	Transitional	I

S. No.	HPE No.	Age	Sex	Site	Histological type	grade
207.	1690/ 04	58	M	Cerebellar	Meningothelial	I
208.	1691/ 04	09	M	Temporal	Fibrous	I
209.	1702/ 04	16	M	Intraventricular	Papillary	III
210.	1710/ 04	61	M	Frontal	Secretory	I
211.	1713/ 04	19	F	Occipital	Meningothelial	I
212.	1714/ 04	35	F	Falx	Angiomatous	I
213.	1716/ 04	45	F	Falx	Papillary	III
214.	1718/ 04	45	F	Parietal	Fibrous	I
215.	1732/ 04	19	F	Occipital	Meningothelial	I
216.	1736/ 04	10	M	Spinal cord	Psammomatous	I
217.	1739/ 04	40	M	Parietal	Lymphoplasmacytic	I
218.	1741/ 04	35	F	Spinal cord	Transitional	I
219.	1751/ 04	45	M	Frontal	Meningothelial	I
220.	1754/ 04	40	F	Parietal	Transitional	I
221.	1756/ 04	44	M	Sphenoid wing	Fibrous	I
222.	1759/ 04	55	F	Sphenoid wing	Transitional	I
223.	1773/ 04	43	M	Parietal	Meningothelial	I
224.	1788/ 04	45	M	Orbit	Fibrous	I
225.	1807/ 04	55	F	Spinal cord	Transitional	I
226.	1817/ 04	35	F	Occipital lobe	Transitional	I
227.	1822/ 04	45	M	Lat. Ventricle	Angiomatous	I

228.	1827/ 05	35	F	Occipital lobe	Meningothelial	I
229.	1838/ 05	35	F	L. CP angle	Fibrous	I
230.	1847/ 05	40	F	Sphenoid wing	Angiomatous	I
231.	1876/ 05	53	F	Falx	Metaplastic	I
232.	1878/ 05	40	M	Sphenoid wing	Fibrous	I
233.	1893/ 05	40	M	Posterior Fossa	Angiomatous	I
234.	1901/ 05	38	F	(L) Petrous	Fibrous	I
235.	1904/ 05	19	M	Posterior Fossa	Psammomatous	I
236.	1908/ 05	40	F	Parietal	Meningothelial	I
237.	1910/ 05	30	F	Parietal	Meningothelial	I
238.	1912/ 05	47	F	Frontal	Transitional	I
239.	1914/ 05	19	F	Temporal	Meningothelial	I
240.	1920/ 05	40	F	Cerebello Pontine	Transitional	I
241.	1921/ 05	39	F	Frontal	Angiomatous	I
242.	1955/ 05	42	M	Petrous	Atypical	II
243.	1959/ 05	45	F	CP angle	Transitional	I
244.	1961/ 05	40	F	Temporal	Transitional	I
245.	1974/ 05	40	F	CP angle	Fibrous	I
246.	1975/ 05	45	F	Frontal	Psammomatous	I
247.	1979/ 05	43	F	Suprasellar	Meningothelial	I

S. No.	HPE No.	Age	Sex	Site	Histological type	grade
248.	1982/ 05	52	F	Temporal	Fibrous	I
249.	1984/ 05	52	F	Frontal	Fibrous	I

250.	2001/ 05	55	M	Spinal cord	Fibrous	I
251.	2003/ 05	35	F	Parietal	Transitional	I
252.	2005/ 05	28	M	Falx	Atypical	II
253.	2016/ 05	32	F	Spinal cord	Psammomatous	I
254.	2017/ 05	34	M	Parietal	Transitional	I
255.	2025/ 05	35	M	Parietal	Transitional	I
256.	2033/ 05	47	F	Spinal cord	Psammomatous	I
257.	2035/ 05	12	M	Spinal cord	Meningothelial	I
258.	2038/ 05	30	F	Frontal	Fibrous	I
259. `	2039/ 05	25	F	Spinal cord	Psammomatous	I
260.	2041/ 05	55	F	Ant. Clinoidal	Fibrous	I
261.	2046/ 05	32	F	Parietal	Meningothelial	I
262.	2047/ 05	50	M	CP angle	Psammomatous	I
263.	2075/ 05	37	F	Suprasellar	Transitional	I
264.	2079/ 05	57	F	Parietal	Fibrous	I
265.	2082/ 05	14	F	Spinal cord	Meningothelial	I
266.	5090/ 05	05	M	Falx	Transitional	I
267.	2091/ 05	35	F	Olfactory groove	Transitional	I
268.	2093/ 05	52	F	Falx	Meningothelial	I
269.	2105/ 05	53	F	Frontal	Transitional	I
270.	2126/ 05	40	F	Posterior Fossa	Fibrous	I
271.	2127/ 05	34	F	Temporal	Metaplastic	I
272.	2128/ 05	35	F	Frontal	Meningothelial	I
273.	2160/ 05	30	F	Sphenoid	Meningothelial	I

274.	2162/ 05	50	M	Frontal	Meningothelial	I
275.	2169/ 05	60	M	Occipital	Rhabdoid	III
276.	2174/ 06	41	F	Para sagittal	Fibrous	I
277.	2178/ 06	28	F	Olfactory Groove	Meningothelial	I
278.	2179/ 06	27	F	Olfactory Groove	Metaplastic	I
279.	2188/ 06	57	F	Parietal	Meningothelial	I
280.	2198/ 06	40	F	CP angle	Meningothelial	I
281.	2216/ 06	40	F	Falx	Fibrous	I
282.	2229/ 06	55	F	Frontal	Atypical	II
283.	2249/ 06	60	M	Falx	Transitional	I
284.	2251/ 06	30	F	CP angle	CP angle	I
285.	2255/ 06	27	F	Frontal	Papillary	III
286.	2258/ 06	68	M	Parietal	Meningothelial	I
287.	2260/ 06	49	F	Frontal	Meningothelial	I
288.	2272/ 06	50	F	Parietal	Psammomatous	I

S. No.	**HPE No.**	**Age**	**Sex**	**Site**	**Histological type**	**grade**
289.	2278/ 06	21	M	Frontal	Fibrous	I
290.	2279/ 06	40	M	Parietal	Transitional	I
291.	2284/ 06	60	F	Frontal	Transitional	I
292.	2287/ 06	18	F	Parietal	Rhabdoid	III
293.	2291/ 06	60	MF	Frontal	Meningothelial	I
294.	2292/ 06	45	F	Frontal	Transitional	I
295.	2293/ 06	35	F	Parietal	Psammomatous	I

296.	2295/ 06	45	M	Spinal Cord	Fibrous	I
297.	2296/ 06	55	F	Temporal	Meningothelial	I
298.	2397/ 06	56	F	Frontal	Angiomatous	I
299.	2301/ 06	40	F	Spinal Cord	Psammomatous	I
300.	2308/ 06	40	F	Frontal	Meningothelial	I
301.	2311/ 06	55	F	CP angle	Fibrous	I
302.	2323/ 06	50	F	Frontal	Fibrous	I
303.	2338/ 06	47	F	Falx	Meningothelial	I
304.	2346/ 06	43	F	Parietal	Fibrous	I
305.	2353/ 06	56	M	Falx	Fibrous	I
306.	2356/ 06	40	F	Spinal Cord	Psammomatous	I
307.	2357/ 06	48	F	Olfactory Groove	Meningothelial	I
308.	2361/ 06	65	F	Parietal	Transitional	I
309.	2381/ 06	60	F	Orbital	Meningothelial	I
310.	2389/ 06	40	F	Intra Ventricular	Meningothelial	I
311.	2404/ 06	23	F	Frontal	Psammomatous	I
312.	2406/ 06	64	F	Spinal Cord	Lympho plasmacytic	I
313.	2416/ 06	42	M	Olfactory Groove	Meningothelial	I
314.	2455/ 06	46	M	Falx	Lympho plasmacytic	I
315.	2473/ 06	27	M	Frontal	Fibrous	I
316.	2475/ 06	40	F	Frontal	Fibrous	I
317.	2484/ 06	42	F	Frontal	Transitional	I
318.	2488/ 06	50	F	Spinal Cord	Psammomatous	I
319.	2489/ 06	41	F	Temporal	Fibrous	I

320.	2493/ 06	55	F	Falx	Transitional	I
321.	2513/ 06	60	M	Parietal	Micro cystic	I
322.	2514/ 06	31	M	CP angle	Atypical	II
323.	2527/ 06	30	F	Orbit	Lympho plasmacytic	I
324.	2531/ 06	52	F	Spinal Cord	Psammomatous	I
325.	2524/ 06	47	M	Clivus	Chordoid	II
326.	2534/ 06	44	F	CP angle	Transitional	I
327.	2540/ 06	45	F	Petrous	Fibrous	I
328.	2551/ 06	60	M	Frontal	Fibrous	I
329.	2556/06	50	F	Petrous	Fibrous	I

S. No.	HPE No.	Age	Sex	Site	Histological type	grade
330.	2558/ 06	63	F	Parietal	Psammomatous	I
331.	2505/ 06	51	F	Sphenoid	Angiomatous	I
332.	2567/ 06	50	M	Spinal cord	Fibrous	I
333.	2569/ 06	20	M	Orbital	Transitional	I
334.	2570/ 06	34	F	CP angle	Fibrous	I
335.	2572/ 06	25	M	Posterior fossa	Meningothelial	I
336.	2641/ 06	65	M	Parietal	Fibrous	I
337.	2652/ 06	27	M	Posterior fossa	Meningothelial	I
338.	2655/ 06	55	M	Frontal	Fibrous	I
339.	2658/ 06	65	F	Falx	Fibrous	I
340.	2667/ 06	26	F	Parietal	Fibrous	I
341.	2676/ 06	41	F	Parietal	Fibrous	I

342.	2680/ 06	45	F	(L) CP angle	Fibrous	I